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Dental Defects in Rats on Defective and Deficient Diet—*Grieves*

Some Important Factors Which Influence Occlusion—*Monson*

Diagnosis of Latent Infection About the Jaws—*Goadby*

The New Recruits—*Editorial*

National Meeting
at
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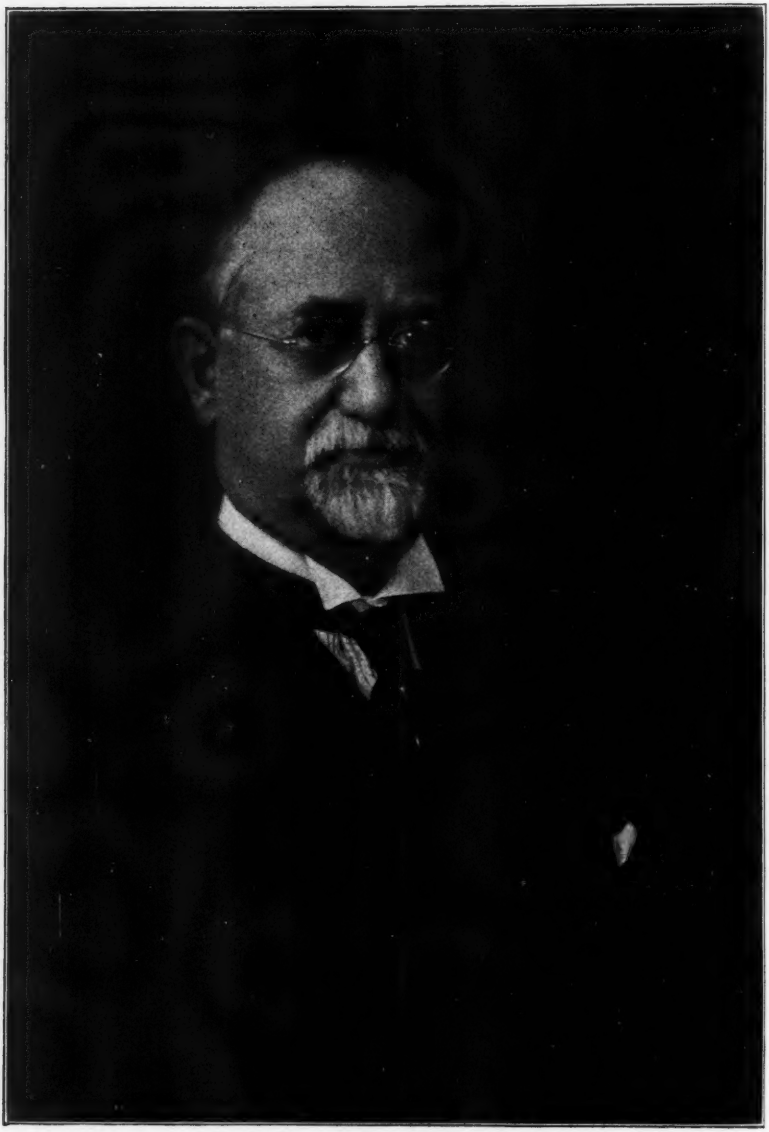

GEORGE H. WILSON, D.D.S.
1855-1922

Like flower bloom and fragrant breath
His life survives the touch of death

THE JOURNAL
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Original Communications

A PRELIMINARY STUDY OF GROSS MAXILLARY AND DENTAL DEFECTS IN THREE HUNDRED RATS ON DEFECTIVE AND DEFICIENT DIET[1]

By CLARENCE J. GRIEVES, D.D.S., Baltimore, Maryland

(Read before the Englewood Dental Society, Chicago, Illinois, November 8, 1921)

THE earliest investigations of diet were concerned with its purity, and contamination was sought by chemical methods; these were applied to determine the purity of protein, carbohydrate and fats. If these were present, together with recognized inorganic salts, in sufficient amounts to furnish energy requirements of the body, the diet was satisfactory.

Dietitians thought in chemistry and spoke in calories and it was believed that these were elementally adequate for life maintenance over many generations.

A dinner consisting of steak, bread made without milk, butter, potatoes, peas and beans, gravy, a flavored gelatin and coffee, appears to constitute a satisfactory meal, for it offers variety and paliability, and meets the requirements of the food chemist and dietitian, but it would not promote health in an experimental animal over a very long period.

An adult rat, when placed upon such

[1]A part of this paper was prepared in the Hunterian Laboratory, Department of Pediatrics, Johns Hopkins University, in conjunction with Drs. E. V. McCollum and Nina Simmonds and E. N. Kinney of the laboratories of the Department of Chemical Hygiene and Public Health, Johns Hopkins University; it is appearing in the *Johns Hopkins Bulletin*, entitled, "The Relation of Nutrition to Tooth Development and Tooth Preservation — A Preliminary Study of Gross Maxillary and Dental Defects in Two Hunderd and Twenty Rats on Defective and Deficient Diets."

a diet, begins to hurry thru life, developing senile changes at double the rate observed in a well-nourished animal. These rats do not litter normally nor fully; they do not succeed well in nourishing offspring and they die fairly early. The young, if restricted to the parents' diet, are never their equal physically, and have increasing trouble with their young; they live a shorter life and all groups are subject to infections and abnormalities unknown to their ancestors. With every generation, weaklings predominate and the strain soon dies out.

Very evidently this diet is defective, but its deficiencies were not known until 1910, when E. V. McCollum (8) and his associates applied biological methods to food analysis. The source of the diet and the minute percentage and relation of its various factors, it was discovered, were of more importance than its chemical anaylsis.

By this procedure each dietary factor, in each of the more important foods is studied separately by slight additions to a known diet, and the extent to which two or more of these enhance nutritional values is determined by reactions in animals, as these mutually supplement known deficiencies (8).

This method has been accepted by other workers and to such an extent has it been developed that, at present, a classification of foods on a biological rather than on an energy production or chemical basis can be interpreted with assurance.

Such is the procedure in the colony of 1,200 rats in the laboratory of the Department of Chemical Hygiene and Public Health, Johns Hopkins University; some 6,000 feeding experiments have been conducted, and volume of experimental data published, to which those interested are referred. It is from these reports (8, 12, 9, 11, 13, 10, 14) that the writer attempts to condense certain basic principles.

In animal experiment, a satisfactory diet might be defined as one which will promote vigor, well-being and longevity, over many generations. In well-nourished rats no nervous disturbance nor departure from normal life habit, no variation in fertility and reproduction, and no difficulty with the young, nor early senility, should occur.

The essentials of an adequate diet for man have been abundantly proved. They are protein of good quality and quantity, that is, the protein must furnish sufficient of the eighteen or twenty amino-acids essential to construction of tissue protein. It must contain a source of the sugar glucose; this may be starch, dextrin or cane, malt, or milk sugar. It must contain mineral elements in appropriate combinations; these are sodium, potassium, calcium, magnesium, chlorine, iodin, phosphorus, iron and sulphur. All of these can be utilized as common mineral salts except sulphur, which must be supplied in the form of the amino-acid cystin, contained in most proteins. In addition, three substances of unknown chemical composition, which we designate collectively as vitamines, and specifically as fat-soluble A, water-soluble B and water-soluble C, are essential.

There is no experimental evidence in support of the view that man or animal actually requires fat in food; apparently it is synthesized by body tissues; at present it is impossible to introduce fat-soluble A into the diet without some fat, so that the matter is not settled.

The relationship of the inorganic elements is most important for normal function of protoplasm and in the proper dietary balance for growth and function. Even slight differences between the calcium, sodium, and chlorine contents of certain common foods is sufficient to de-

termine whether or not growth in an animal will take place. The notable relation of calcium and phosphorus to fat-soluble A, as illustrated in bone pathology and blood calcium, needs only to be mentioned.

Animal experimentation has shown the greatest difference in biologic value of protein from different sources, depending upon the yield of amino-acids or digestion products. If the yield permits easy conversion into tissue protein, that protein is of high food value, since these acids cannot be synthetically produced by the tissues.

Two opposing views exist regarding the amount of protein intake necessary to produce the best results. The low-protein advocates believe in just enough protein consumption for "specific dynamic action" of protein to stimulate metabolism. They insist that a large protein intake furnishes pabulum for excessive growth of intestinal putrefactive bacteria, resulting in absorption of toxic products of the degradation of certain amino-acids in quantity sufficient for tissue damage.

The high-protein advocates regard the consumption of liberal amounts of meat as essential to vigor and aggressiveness. They point to the fact that the races which predominate in history have been large consumers of meat protein.

Among all progressive peoples, while meat is an important dietary factor, it is usually supplemented by dairy products, and it is the correction of muscle deficiencies by these, rather than the ingestion of much meat, which is so important.

A liberal intake of protein of good quality from meats, seeds, and dairy products will often promote health and vigor, when all other factors fail.

The treatment of tuberculosis by "heavy" protein diets of milk and eggs is notable. In the rat, normal growth is secured on diets containing 9 per cent protein of high biological value; if it falls below this percentage in excellence or quantity, growth and reproduction are far below normal expectation. Rats thrive on high protein diets and it cannot be assumed that such would generally be safe for man; for some of the intermediate compounds, produced by catabolism of the amino-acids are physiological abominations. The evils attributed to excessive protein consumption, however, often result from their character and from other faults in dietary balance rather than from protein.

The following shows the biological value of proteins, the best is placed first, and the series descends: Beef kidney; wheat; milk, liver; muscle cuts, barley, rye; maize, oats; soy beans, navy beans, peas.

Animals do not remain at the optimum when there is a close approximation of the physiological minimum of any dietary factor; *it is the equalization and balance of all factors which make for health.*

The American diet always so nearly approaches this plane of physiological minimum that any slight dietary deficiency on the one hand, or increased physical or nervous stress on the other, will induce nervous or deficiency disease, or by decreasing resistance it may lay the patient open to any type of infection.

There is nothing original in the thought that common mouth organisms, of such low virulence that they evidently failed to invade our ancestors or to attack the present generation, if in health, and which are resisted by the aged, who have overcome many pathogenic organisms, are today producing such an increasing percentage of chronic disease and death as to be appalling. *That a liberal and correctly balanced intake of all dietary factors may combat these ills is possible.* It is the hope of the modern dietitian to discover such combinations.

It has been found that parts of plants,

functioning as storage tissues, seeds, tubers, and fleshy roots have remarkably similar dietary properties and shortcomings. Notwithstanding the great differences existing between legumes and seeds, as wheat, the pea and bean, the potato and turnip, they have nearly the same dietary values. All cereal grains, legume seeds, tubers and edible roots are deficient in three important dietary factors; all contain protein of poor quality, too little calcium, sodium, and chlorine, and little fat-soluble A. Quite the opposite, leaves of plants, which consist of actively functional protoplasm, contain all of these. The greatest contrast exists between the two, for palatable leaves and grasses alone are complete foods for animals with gastrointestinal tracts so complicated as to absorb enough digestive products for their energy requirements.

Leafy plants and seeds thus mutually supplement each other's deficiencies, both with respect to the inorganic moiety and in fat-soluble A.

A similar parallel between function and dietary properties exists in highly specialized muscle tissue and actively metabolizing glandular tissue. The dietary properties of muscular tissue are identical with the seeds and tubers, except that it is richer in protein. It lacks calcium, sodium, chlorine and the essential vitamines A, B and C. Liver, kidney, and the glandular organs contain fat-soluble A in the interstitial fats. They contain all of the complexes for construction of living tissue when supplemented by certain salts and carbohydrates, such as starch. Animal tissues, kidney, liver and muscle, are superior to milk in making good any protein deficiency in seeds and tubers; but milk is so constituted as to greatly improve the inorganic content, the quality of the protein, and to increase the fat-soluble A.

The popular mind is very enthusiastic regarding the spectacular deficiency diseases resulting from a lack of the essential food factors A, B and C. Scurvy, involving the mouth, intestines, the periosteum and bones, resulting from deficient antiscorbutic substance, water-soluble C, beriberi or polyneuritis, resulting in paralysis, from the absence of the antineuritic, water-soluble B, and xerophthalmia, with loss of sight and malnutrition from the lack of anti-xerophthalmic substance, fat-soluble A, are all tragic and most impressive diseases, which, if not properly treated, result in death.

It is our belief that minor phases of these deficiency diseases exist, which are not, as yet, clinically apparent and that many of these may involve the maxillae, teeth and attachments and oral mucosa and glands. It is to these that we would direct your attention, for dentists fortunately rarely come in contact with the major expressions, but evidences of slight deficiency disease not only in vitamines but in all basal dietary factors are continually under our observation.

The distribution of the vitamines is as follows:

Water-soluble C is abundant only in fresh vegetables, salads, fruits, or fresh milk, from cows in pasture; it is absent from dried or cooked food.

Water-soluble B is widely distributed in common foods but lacking in polished rice, sugars, starches, and animal vegetable fats and oils. It is not damaged by ordinary cooking and exists in small quantities in white flour, degerminated meals and muscle cuts, while whole seeds, tubers, leafy foods, milk, eggs, and glandular organs are amply supplied.

Fat-soluble A exists in greater abundance in butter, egg yolk, and in interstitial fats from liver and kidney than in any other food; edible leaves are its next important source, while seeds, tubers and roots contain but little of it.

Vitamine deficiencies have recently overshadowed long recognized dietary essentials, *and there is no basis in fact for considering them any more important*

than protein or mineral or other basal elements in the diet.

In animal experiment, the rat requires, in addition to the common dietary complexes, only fat-soluble A and water-soluble B; it synthesizes C; while other species as man, swine, dogs, monkeys and guinea-pigs require all three.

The guinea-pig normally takes a diet rich in succulent vegetables, which produces bulky, easily eliminable feces. The rat, and swine, as well as man, thrive on certain diets, which leave little indigestible residue. *Such special requirements in guinea-pigs make it next to impossible to compare this species with man or the rat in similar dietary studies. Experimental data obtained with guinea-pigs must be used with caution in conclusions as to the etiology of human scurvy, and we might add other dental and maxillary diseases.*

After extensive inquiry, McCollum and his associates have determined that but three diets succeed in animal nutrition:

1. *The carnivorous type diet.* This diet succeeds if not restricted to muscle meat, which is the usual misconception of a carnivorous diet, for young animals cannot grow or remain healthy on muscle as the sole food. When the entire animal is fed, blood, bones, kidney, liver and glandular tissues, rats do well. This is the truly carnivorous diet of primitive Eskimos and Indians, supplemented occasionally by leaves and corn.

The primitive Eskimos have few dental defects; their diet is rich in protein and fat, but fairly satisfactory in other factors, as all blood, marrow, and glandular organs are saved and eaten, and such soft bones, as ribs are chewed. In northern Alaska the younger generation of Eskimos, near the trading stations, who live largely upon cereal grain flours, canned products and muscle meats, suffer from all types of dental disease. That oral deterioration in these primitive races occurred simultaneously with mondification of diet induced by contact with the white man's grocery store is significant, for this diet is usual in the United States. Similar conditions exist among the inhabitants of Newfoundland and Labrador, who eat patent flour bread, fish, salt meats, tea, raisins, with few vegetables and who also suffer from beriberi, xerophthalmia and night blindness. Phases of these diseases also occur in lumber camps among all races confined to restricted diets. The condition of the health of the non-citizen Indian, who subsists solely on government rations as compared with the good health of the Indian who adds to these a garden and keeps a cow, is an interesting illustration.

2. *The seed and leaf type diet.* It has been found possible to supplement the seed, tuber, root and occasional muscle meat diet with liberal quantities of leafy vegetables, securing a fairly satisfactory result. This is the diet of the oriental, who too frequently is compelled to eat that he may live. It is difficult to persuade our people, when other more attractive foods are available, to consume such quantities of greens as are necessary dietary supplements. Polyneuritis, resulting from a diet of fish and polished rice is common in the orient, as is xerophthalmia; the latter is cured, significantly enough, by a diet of liver, just as night blindness is cured by feeding cheese and milk.

3. *The seed, muscle and milk diet.* This type consists of cereals, legume seeds, tubers and fleshy roots, with or without meats, supplemented with liberal amounts of milk. Milk is rich in protein, mineral salts and fat-soluble A, in which seeds and muscles are poor. Milk and leafy vegetables are well termed "the protective foods."

The pastoral peoples, past and present, of Central Asia and Eastern Europe, use quantities of sour milk in the diet. If physical well-being as exemplified in excellent development, courage, and en-

ergy be the first requisite of a good diet, these tribes are excellent examples. Under many names fermented milk is used extensively as food, supplemented by fruits. The physical development of such people is unusually good, and length of life exceptionally long, as instanced in Arabia and Bulgaria.

The theory that some specific action of lactic acid organisms in sour milk diets prevents intestinal putrefaction, must now be interpreted by modern biological dietary tests. It is the correction of deficiencies of the seed muscle diet by the inclusion of quantities of the protective food (milk) which greatly increases health and vigor, rather than the bacteria, which are of secondary importance.

It is said that prior to 1850 dental caries was rare in Iceland and that no dental defects occurred in ninety-six skulls from the ninth to the eighteenth centuries. The diet of the Icelander prior to 1850, who lived by fishing and keeping herds, consisted of milk, mutton, fish and fowl, with oat cake, carrageen moss, a few potatoes and turnips. Dental deterioration is said to have begun after 1850 with the regular importation of flour and grocery store products.

Milk is undoubtedly one of the dietary factors which lead to longevity. The composition of human and cow's milk depends upon dietary complexes, which can be secreted by the mammary glands from the blood. A cow fed on grain alone cannot synthesize very essential protein and salts, as calcium and phosphorus, or fat-soluble A, from diets where these sparingly exist; she must have digestible leaves and grasses, where they do exist.

This is equally true of the human mother, thousands of whom are attempting to nurse infants on diets consisting of wheat flour, degerminated cornmeal, polished rice, potatoes, muscle cuts, starches, together with reserve animal fats, as suet and lard, sugar, tea and coffee. This diet is deficient in inorganic salts, protein and fat-soluble A, which are such important factors in good milk. *Such diets produce inferior milk in both cow and mother and lead to grave disorders in the very young, and profoundly affect the teeth thruout life.*

The prevalence of rickets in babies and young children suffices to convince even the most skeptical *that we are in ordinary practice actually feeding our infants and children so as to induce malnutrition of this type in very numerous cases.*

The history and importance of the dairy industry, as a factor for safety, in the population of the more progressive nations cannot be too strongly accented, for it applies to the health of the adult as well as to the mother and child. In this country, with an increasing tendency to cultivate grains for commercial reasons instead of truck gardens and dairy farms, the value of dairy products as corrective dietary factors cannot be overestimated.

The very important question as to the relation of all sugars, particularly cane sugar, arises at this time. The tremendously increasing dietary ratio of sugar is undoubtedly doing much harm if in no other way than by satisfying quickly the energy demands and appetite and displacing from the diet more valuable foods.

It is not possible as yet to report on the relative value of sugars, nor their effect upon teeth from these laboratories as no cane sugar occurred in the fifty-seven diets studied.

Of the numerous diets employed for the study of the relation of certain nutritive principles to bone pathology, fifty-seven were selected as affording data which would be especially worth while for the study of the relation of the diet to the development of oral structures. These were so constituted that the effect of small variations in quality of several

dietary factors on the maxillae, teeth and attachments could be interpreted.

It is now possible to study the dietary factors in the relation to tooth development and to permanency of these structures under circumstances far more favorable than hitherto because we now are in possession of much knowledge concerning the nutritive needs of the animal, and also of the dietary properties of a large number of our more important foodstuffs. Furthermore, the specific effects of faulty diets of many kinds have been carefully observed, and in the case of rickets we have an extensive knowledge of the chemical causes underlying a pathological syndrome of great significance.

For more than twelve years we have maintained our breeding stock of rats on a monotonous dietary regimen, which experience had taught us was more satisfactory in inducing vigorous growth, high fertility, and success in the rearing of vigorous young than could be attained by feeding table scraps, dog biscuit, bread and milk or a mixture of grains, forage plants and of foods of animal origin, where a freedom of selection was permitted the animals. With this diet we have been so successful that we have steadily improved our animals from year to year, not alone thru uninterrupted nearly optimal nutrition, but also thru rigid selection, year by year, of the most perfect specimens for renewing the breeding stock. We have employed animals from this stock as a standard of excellence with respect to dental structures with which others of the same stock (their own progeny), but reared on diets which deviated in some one or more factors from the ideal relationship or composition essential for optimal nutrition

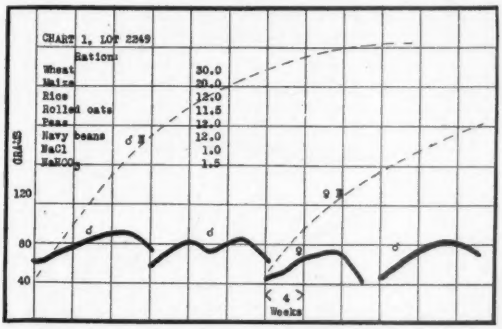

Chart 1.—Lot 2249 was fed from weaning time on a diet of cereal grains, legume seeds (peas, beans) supplemented with sodium chloride and sodium bicarbonate. The latter salt was added because the cereals all contain an excess of acid-forming elements (chlorine, phosphorus, sulphur) over basic elements (calcium, magnesium, sodium, potassium) and the amount added was sufficient to make the diet as a whole slightly alkaline.

The chief fault in this diet was lack of calcium. According to our studies this mixture of cereal grains and legume seeds contained only about one-fifteenth of the optimal calcium to meet the needs of the growing rat. It contained only about half the requirements of fat-soluble A for this species. The protein content was somewhat below the optimum, both in amount and quality, and the diet could be enhanced by the addition of phosphorus, altho the deficiency in this element was not marked.

On this diet the animals grew very little and declined and died after about three months. They were short, stocky, and rough-coated. Their bones were poorly developed.

TABLE I. PERCENTAGE COMPOSITION AND SOURCES OF THE EXPERIMENTAL DIETS EMPLOYED IN THESE STUDIES

No. of Ration	No. of Chart	Wheat	Maize	Polished Rice	Rolled Oats	Peas	Navy Beans	Flaxseed Oil Meal	Casein	Beefsteak	Liver	Milk Powder	Butter Fat	Cod Liver Oil	$CaCO_3$	NaCl	$NaHCO_3$	KCl	Dextrin	Remarks
2249	1	30.0	20.0	12.0	11.5	12.0	12.0	...	.....	.....	.....	.....	.....	....	.....	1.0	1.5	.....	.....	Low calcium, low fat-solution A, 12.3 per cent of poorly constituted proteins
2188	2				40.0	.....	.....	.....	.....	4.1	.....	.....	3.0	....	1.5	1.0	.....	.....	50.4	Low protein (9.0 per cent) of relatively low biological value
2667	2	.....	.....		40.0	.....	.....	8.3	.....	.....	.....	.....	.....	....	1.5	1.0	.....	.....	49.2	Low protein (9.0 per cent) of low value. Low fat-soluble A
2769	3	30.0	10.0	10.0	10.0	10.0	10.0	.....	10.0	.....	.....	.....	10.0	.....	.....	.....	.....	.....	.....	Low calcium diet
2581	3	.....	.....	.....	.....	.....	.....	.....	10.0	.....	20.0	.....	3.0	....	.....	1.0	.....	1.0	65.0	Low calcium diet
2883	3	.....	.....	.....	.....	.....	.....	.....	10.0	.....	20.0	.....	.....	2.0	.....	1.0	.....	1.0	66.0	Low calcium diet
2774	4	25.0	20.0	10.0	10.0	9.5	9.5	.....	10.0	.....	.....	.....	2.0	1.0	3.0	.....	.....	.....	.....	Twice the optimal calcium content
2883	4	25.0	10.0	9.5	9.5	9.5	9.5	.....	10.0	.....	.....	.....	.....	7.0	3.0	1.0	.....	.....	.....	Twice the optimal calcium content, with high cod-liver oil
2779	5	30.0	20.0	11.5	11.5	10.0	10.0	.....	.....	.....	.....	.....	3.0	.....	3.0	1.0	.....	.....	.....	12.3 per cent of protein of relatively poor quality
2775	6	25.0	20.0	9.5	9.5	9.5	9.5	.....	10.0	.....	.....	.....	3.0	.....	3.0	1.0	.....	.....	.....	Twice the optimal content of calcium
2767	7	30.0	19.5	9.5	9.5	9.5	9.5	.....	10.0	.....	.....	.....	.....	1.0	0.5	1.0	.....	.....	.....	Twice the optimal amount of calcium
2865	10	20.0	10.0	10.0	10.0	10.0	10.0	.....	10.0	.....	.....	10.0	10.0	.....	.....	.....	.....	.....	.....	About one-third the optimal content of calcium. Slightly less than optimal content of cod liver oil
2406	11	20.0	19.5	9.5	9.5	9.5	9.5	.....	.....	10.0	.....	10.0	.....	.....	.....	1.0	.....	.....	1.5	Low calcium diet
3020	11	25.0	19.5	9.5	9.5	9.5	9.5	.....	10.0	.....	.....	5.0*	.....	.....	.....	1.0	.....	.....	1.5	Low calcium, low fat-soluble A
2766	12	20.0	15.0	9.5	9.5	10.0	10.0	.....	10.0	.....	.....	5.0	10.0	.....	.....	1.0	.....	.....	.....	Low calcium, low fat-soluble A
2838	13	29.0	19.5	9.5	9.5	9.5	9.5	.....	10.0	.....	.....	.....	.....	1.0	1.5	1.0	.....	.....	.....	Low calcium diet
																				Optimal calcium, slightly below optimal content of fat-soluble A

*Krystalak.

NOTE—Low calcium signifies a content of calcium derived from the natural foods included in the diet.

TABLE II. PERCENTAGE COMPOSITION AND SOURCES OF EXPERIMENTAL DIETS

No. of Ration	No. of Chart	Bolted Flour	Rolled Oats	Cornmeal	Whole Milk Powder	Potato	Polished Rice	Peas	Navy Beans	Red Beets	Yellow Turnips	Casein	Beefsteak	Dextrin	NaCl	Cod Liver Oil	Remarks
2763	9	20.0	.....	10.0	10.0	23.0	.....	8.0	8.0	5.0	5.0	10.0	.....	.....	.....	1.0	Low calcium, and below the optimum in fat-soluble A. Deficient in sodium chloride
2408	8	20.0	9.5	19.5	10.0	.....	9.5	9.5	9.5	.....	.....	.....	10.0	1.5	1.0	.....	Low calcium, and below the optimum in fat-soluble A. Approximately the optimal content of sodium chloride

might be composed. An excellent criterion of the degree of success which we have attained in the feeding of our breeding stock is the nearly complete absence of dental lesions in most of the animals from these controls, even tho they were twelve to fifteen months old or nearly halfway thru the extreme span of life of this species.

The stock ration consists of a mixture of wheat, maize and rolled oats, 30 per cent each, flax-seed oil meal 10 per cent, whole milk *ad libitum*, fresh cabbage or carrots twice a week. The seed mixture was ground together so finely as to preclude the possibility of the animals picking out and eating the components separately.

The experimental diets employed in these studies are tabulated and described in Tables I and II. For detailed discussions of these diets reference should be made to the charts.

A brief resume of dental etiological theories, past and present, is necessary if we are to approach the study of the etiology of dental and maxillary defects in animal experiment intelligently.

The major dental diseases, abrasion, erosion, caries, gingival lesions and all resultant sequelae have been associated

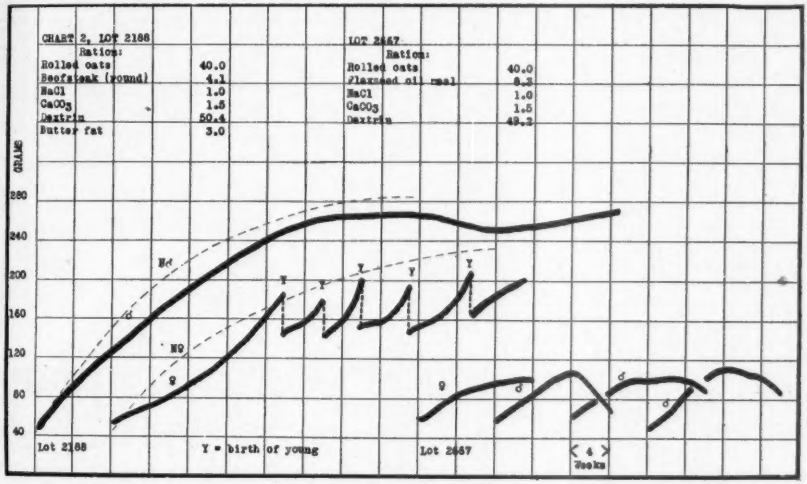

Chart 2.—Lot 2188 had from weaning time a diet containing but 9 per cent of protein of fairly good quality. There were no inorganic deficiencies since the beefsteak contained phosphorus in liberal amounts and calcium carbonate was added in quantity sufficient to make the calcium content near the optimum. The 3 per cent of butter fat served as a source of fat-soluble A and furnished enough of this factor to support good growth. The animals grew fairly well but never reached the maximum size. Their fertility was high but practically all the young died. Their coats became rough and they looked old at an early age. The cause of this lay in insufficiency of the protein content of the diet.

Lot 2667.—The diet of these animals contained 9 per cent of protein of rather poor quality. It was very low in fat-soluble A and in phosphorus but contained about the optimal amount of calcium. On this diet ophthalmia developed in five to eight weeks because of the lack of fat-soluble A. The animals grew but little and died after about three months. Their coats were never sleek and they presented a poorly nourished appearance. The bones of these rats were poorly developed.

TABLE III. PERCENTAGE INCIDENCE OF DENTAL DEFECTS IN RATS RESTRICTED TO DIETS OF DIFFERENT CHARACTERS

No. of Diets Studied		Caries-like Defects in Molar Group	Loss of Attaching Tissue and Teeth, or Both, in Molar Group	Fractures in Incisors and Molars	Pulp Exposure and Osteodentin in Incisors	Overgrowth in Incisors	Maxillary and Mandibular Damage	Percentage of Total Damage		Calcium per 100 Grams Ration	Phosphorus per 100 Grams Ration
1	Stock ration No defects of a significant magnitude. Our breeding stock of rats has been confined to this diet thru a period of twelve generations without any deterioration in vitality. During this interval they have improved physically because of the selection of exceptional individuals for breeding	0.0	0.00	0.02	0.00	1.10	0.00	0.36			
7	Diets low in calcium but rich in fat-soluble A A diet typical of this group is described in Chart 10, Lot 2865. Factors other than calcium in this diet were of nearly optimal quality	1.18	20.81	2.59	19.08	8.31	19.04	11.66	Lot 2865	0.1348	0.4110
6	Diets containing excessive calcium, and with a satisfactory content of fat-soluble A In this group are included animals fed diets similar to or identical with those described in Chart 5, Lot 2779, and Chart 6, Lot 2775. The latter differed from the former in that it contained 10 per cent of casein, and therefore furnished 0.08 grams of phosphorus per hundred grams of ration more than did diet 2779. This modification in the phosphorus content of diet 2775 improved the growth curves, and resulted in better nutrition than would the same food mixture without the added casein. This difference was not due entirely to the added phosphorus, but in part at least to the approved amino-acid supply resulting from the addition of casein. Notwithstanding these differences in the two diets, the effects on the dentition of the animals was so similar that the six diets corresponding to these types are grouped together here. We are now studying in detail the many specific problems which are suggested by these preliminary results	1.73	33.38	3.28	0.00	0.00	12.50	8.48	Lot 2779 Lot 2775	1.2530 1.2482	0.3459 0.4053
5	Diets low in calcium, low in fat-soluble A, and proteins of relatively low biological value Growth of young rats on diets of this class is very poor (Chart 1, Lot 2249). All diets of this class in which the proteins are derived solely from cereal grains and legume seeds are below the optimal in the quality of the amino-acid mixture which they yield. This means that the proteins are not so effectively transformable into body proteins as is possible with proteins of better quality	2.88	35.50	4.70	37.50	17.85	45.20	23.93	Lot 2249	0.0216	0.3631
6	Diets containing excessive calcium, and containing several percentages of cod-liver oil, or of cod-liver oil and butter fat Diets 2774 and 2933, Chart 4, are typical of those fed to animals classed in this dental group. The protein content was of good quality, and was abundant in amount. The calcium content was about twice the optimal. (The optimal content is about 0.64 per cent calcium, equivalent to about 1.5 per cent of calcium carbonate added to a mixture of cereal and legume seeds.) The content of fat-soluble A in these diets was very liberal in terms of the nutritive requirements of the rat	3.00	16.70	4.48	4.16	0.00	6.93	6.21	Lot 2774 Lot 2933	1.2482 1.2476	0.4053 0.3982

7	Diets low in fat-soluble A and low in phosphorus. They contained 9 per cent of protein of relatively low biological value A typical example of diets of the type under consideration is Lot 2667, Chart 2. They were so deficient in fat-soluble A that the animals developed xerophthalmia within a few weeks and their lives were cut short. The skulls examined were, therefore all of animals which were chronologically young, altho they showed physiological deterioration. This is reflected in the high percentages of molar defects in animals but three to five months old....	3.12	19.74	8.48	5.00	10.00	18.50	10.80	Lot 2667	0.6668	0.2214
6	Diets low in calcium, but otherwise well constituted The fat-soluble A content was abundant. The protein was in all cases of very good quality. The calcium content was so low as to cause in some groups stunting of growth, and early deterioration and death (see Chart 3, Lots 2769, 2581, 2883, and Chart 12, Lot 2766). In other diets small additions of milk powder aided the calcium content sufficiently to permit the animals to grow at a subnormal rate. Nothwithstanding their capacity to grow, the animals on this latter class of diets (Chart 10, Lot 2865, and Chart 11, Lot 2406), were physically inferior to animals on better diets. The high incidence of molar defects in these animals is an index to their nutritional instability	3.65	57.90	4.45	14.57	6.25	39.90	20.12	Lot 2769 Lot 2581 Lot 2883 Lot 2766 Lot 2865 Lot 2406	0.0480 0.0010 0.0010 0.0904 0.1348 0.1356	0.3868 0.4000 0.4000 0.3929 0.4110 0.4301
7	Diets low in protein (about 9 per cent) of relatively low biological value. They were not far from the optimum in their content of fat-soluble A. An example of the diets in this group is given in Chart 2, Lot 2188. The only pronounced defects in these diets was in the protein moiety. The percentage of dental defects in rats restricted to these diets was so high that we have undertaken a comprehensive study of this factor in relation to tooth development and tooth vitality	4.74	9.47	0.08	20.82	7.72	19.71	10.91	Lot 2188	0.6371	0.1753
7	Diets low in calcium, and below the optimum in fat-soluble A The calcium content of the diets in this class stood intermediate between those which derived all their content of this element from cereal grains and legume seeds, and those in which approximately the optimal content of calcium was added in the form of calcium carbonate, viz., 1.5 per cent $CaCO_3$. Typical examples of these diets are shown in Chart 7, Lot 2767 and Chart 8, Lot 2763. All factors other than protein and fat-soluble A were well constituted....	5.21	7.70	2.88	2.14	3.38	0.00	5.25	Lot 2767 Lot 2763	0.2503 0.1817	0.4217 0.3713
6	Diets low in calcium, and with fat-soluble A distinctly below the optimum The milk powder contained in these diets was not sufficient to introduce enough calcium to do more than enable the animals to grow at a rate which would seem to indicate fairly good nutrition. Actually we know, from the results of our observations of the life-histories and reproductive capacity of such animals that they were in a state of nutritive instability. Their span of life is distinctly below the normal, and their infant mortality was high. The young were inferior in appearance and the families tended to die out within two or three generations. The percentage of dental defects was high	5.26	14.94	1.88	37.45	11.10	13.83	14.00	Lot 2406 Lot 2408 Lot 3020	0.1356 0.1363 0.0936	

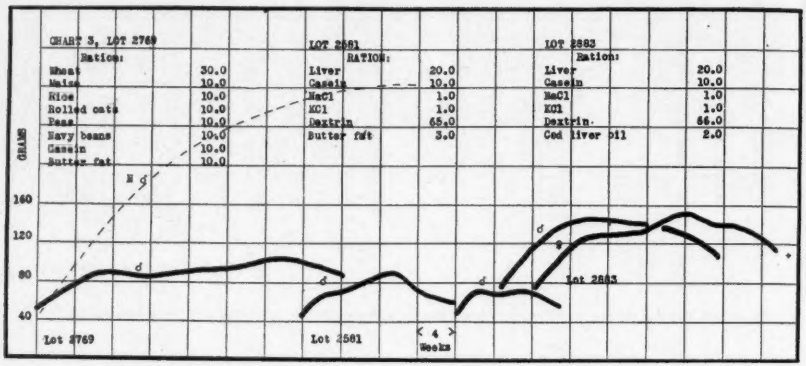

Chart 3.—Lots 2769, 2581 and 2883 may be considered together since the only deficiency in any of them was lack of calcium. Casein enhanced the quality of the vegetable proteins in Lot 2769. In Lots 2581 and 2883 the casein and liver sufficed as an excellent source of this factor. Butter fat and cod liver oil furnished fat-soluble A. All other factors except calcium were satisfactorily constituted. When suitable amounts of calcium carbonate are added to any one of these diets, good growth and high fertility are secured. Without calcium additions but little growth was possible and no young were secured in any of the groups in each experiment. The curves in these charts are those representing the growth of a typical individual in a group.

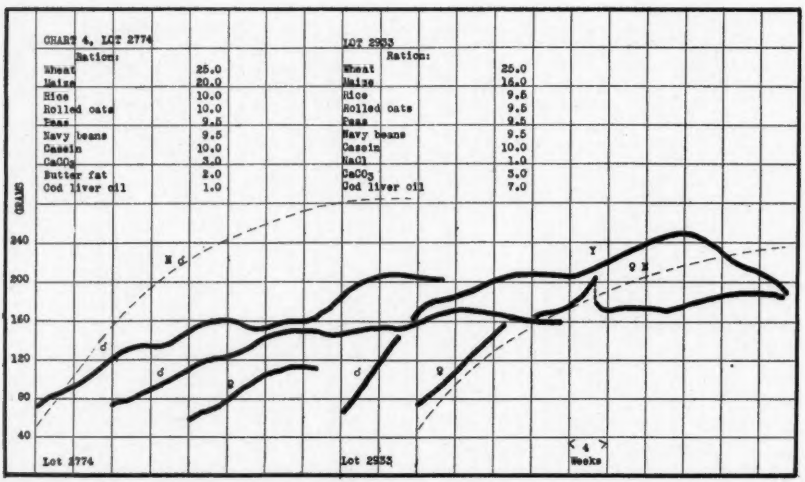

Chart 4.—Lots 2774 and 2933. These two groups had diets which were well constituted except that each contained about double the optimal amount of calcium. For this reason their growth was somewhat depressed and their fertility was interfered with. They had poor coats and did not look well nourished.

with dietary and metabolic disturbance in dental literature since its inception.

The earliest conception of the causes of oral and dental disease may be divided into the systemic or internal, and interstitial, and the external or environmental factors.

Hippocrates (7) taught that dental caries was due to "stagnation of depraved juices in jaws and teeth," aggravated by accumulation of food debris.

Galen (7) believed that disturbance of nutrition produced vicious humors of the blood and caused teeth to decay.

John Hunter (7) maintained that teeth were destroyed by internal inflammation and not by external influences.

The more modern etiological hypotheses may be divided into the traumatic, inflammatory, infectious, toxic, chemical, chemico-micro-organic and nutritional (5, 3, 17).

Dental defects were conceived to be caused externally by trauma or by decalcification of enamel from acid foods, drinks, and medicines. After the discovery of fermentation, it was believed that micro-organisms, which converted starches and sugars into acids, existed in the mouth and decalcified enamel. Other types of bacteria were said to invade and decay dentin by both fermentative and putrefactive processes (17).

The saliva was thought to be acid from acid foods or from acid-forming bacteria. Its acidity was also regarded as an expression of lowered blood alkalinity. Inorganic waste in the saliva, which was deposited gingivally, was known to form salivary calculus. This caused irritation of the gums and was said to induce gingival disease.

This conception of local damage by local secretions was applied to solve the

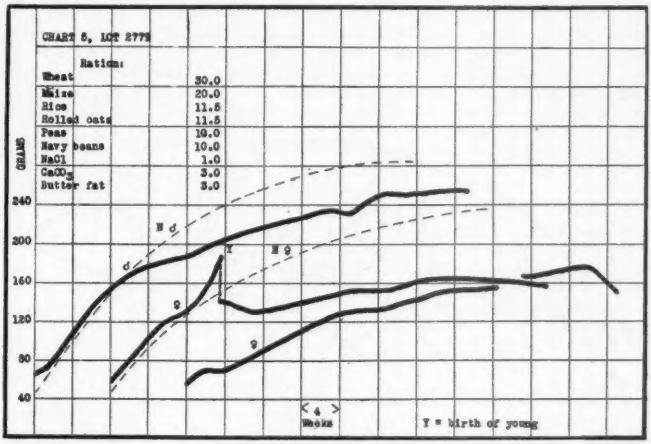

Chart 5.—Lot 2779 had a diet containing double the optimal amount of calcium. The phosphorus content was too low. It should be remembered, however, that a high content of calcium in the diet is tolerated better if the phosphorus content is also high. The content of fat-soluble A was sufficient to enable the animals to grow well, provided all other factors in the diet are properly adjusted, but more of it would have aided the animals in this experiment to tolerate better the excessive intake of calcium and the faulty proportion between this element and the too low phosphorus supply.

These animals aged early, had poor coats and appeared poorly nourished. The fertility was low and all the young born died early. They should be compared with Lot 2775, Chart 6, where the diet is similar except that the latter received 10 per cent of a purified protein, casein.

etiology of dental erosion. The soluble acid calcium and sodium salts, abnormally excreted by the oral mucous glands, were believed to erode cervical enamel.

The various phases of gingival disease culminating in pyorrhea were said to result from external causes, acting from without inward; the theories of "traumatic occlusion" and of specific infectious invasion may be mentioned as examples. But many of the gingivitides were observed to have their origin in the organism itself and to progress from within outward; examples of this type resulted from scurvy and from massive doses of mercury.

Gingivitis was regarded as the result of "auto-intoxication," of "excess of uric acid," and of toxic absorption from the gastrointestinal tract. But essentially the idea was that of deranged metabolism, "sub-oxidation," or defective elimination, rather than that of an improperly constituted diet.

The use of bread made of decorticated and degerminated flour was regarded as one of the causes of dental caries. Upon such a diet, it was stated that expectant mothers suffered much from dental caries and the child's teeth were so poorly calcified as to be subject to early decay.

Proprietary infant foods were believed to produce enamel hypoplasia, defective dentin and general dental dystrophy.

It was claimed that an excessive carbohydrate diet produced caries in both adults and children and an excess in protein was thought to produce pyorrhea in adults.

The endocrine glands, particularly the parathyroid and pituitary, were indicted for causing maxillary dystrophy, malocclusion and dental disease, presumably because of their apparent relation to

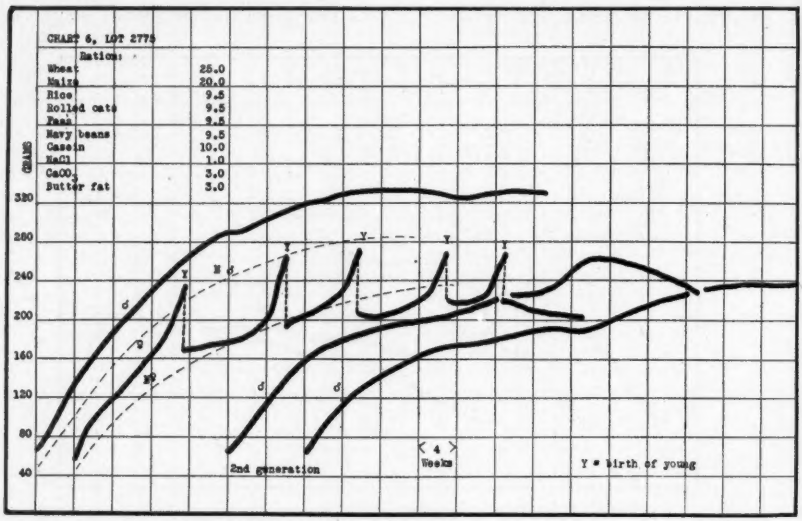

Chart 6.—Lot 2775 had a diet which was well constituted except that the calcium content was too high (approximately double the optimum). They grew well and their fertility was high, but the infant mortality was also high. Several young which were reared, were continued on the diet. Their growth was somewhat stunted. An excessive intake of calcium causes early deterioration. It should be appreciated, however, that the optimal calcium intake for the rat is approximately fifteen times the content of this element contained in the cereal grains.

growth and to calcium and sugar metabolism. The oral conditions found in cretins and acromegalics and in dyspituitarism were cited as examples.

It had long been assumed that dental caries was caused by excessive sugar in the diet. The incidence of caries was said to develop in the same ratio as sugar consumption. It was supposed to increase the blood sugar which, it was claimed, furnished a fermentable media for bacteria in the dentinal tubuli. The salivary glands were said simultaneously to excrete glycogen, which fermented on enamel surfaces. So both locally and systematically, sugar was supposed to attack the teeth.

The literature on scurvy and rickets has been quoted to show that deficiencies in the vitamines, water-soluble B and fat-soluble A, caused caries of bones and teeth and pyorrhea.

The statement that "a clean tooth never decays" was used in oral hygiene propaganda and marked results were reported from the practice of oral prophylaxis by patient and operator. Cleanliness is a relative condition and it is now known from a study of those who sedulously practiced oral cleanliness that caries and gingival disease recur in the so-called "clean mouth."

From this etiological review, we can only conclude that oral and dental dis-

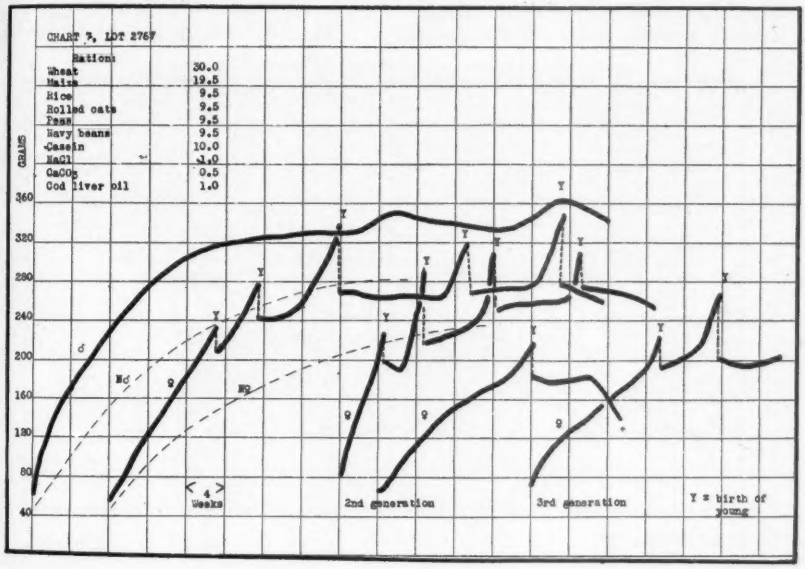

Chart 7.—Lot 2767. This diet was well constituted in every respect except that the calcium content was a little less than half the optimum. The cod liver oil supplied a high content of fat-soluble A and this aided greatly in enabling the animals to tolerate the deficiency of calcium in their diet. We believe, however, from the experience with other experimental diets, that double this amount (2 per cent) of cod-liver oil would have further contributed to their physiological well-being.

Growth was excellent in this group. The fertility was high and the infant mortality was low. Ths are the criteria of good nutrition. The second or third generations of animals reared on this diet showed no definite signs of inferiority.

eases arise from internal, or systemic, and external, or environmental causes, or a combination of such causes.

There is a definite relation of oral types; the temporomandibular articulation, maxillae and occlusion, the tooth form, cusps, roots and attachments, to the form of the gastrointestinal tract and to food habits (21). These must be considered in selecting animals for oral study. *The highly specialized and persistently growing molars of the guinea-pig and its equally specialized gastrointestinal tract are not comparable to those of man, nor is the guina-pig, with its limited diet of roots and grasses, suited to the study of the omnivorous human diet.*

The dog's salivary and gastrointestinal functions are well known, while comparatively little is known of these functions in the rat. The dog's denture is large and accessible, and presents few of the histologic difficulties found in the study of the small molars of the rat. But if dogs are allowed to run, they may obtain supplementary food, thereby disturbing the experimental conditions.

If confined, they may develop nutritional disturbances, deformity and disease. The rat thrives under laboratory conditions, and is therefore the animal of choice for studies of nutrition.

"The (6) rodents include by far the greatest number (nine hundred) and have the widest distribution, of any order of terrestrial mammals." Of the subfamily, Muridae, which included M. Norvegicus (used in these studies) it is said: "This genus is the largest in the whole mammalian class, comprising not less than one hundred and thirty species." Such preponderance is founded in great fertility and adaptability, especially to

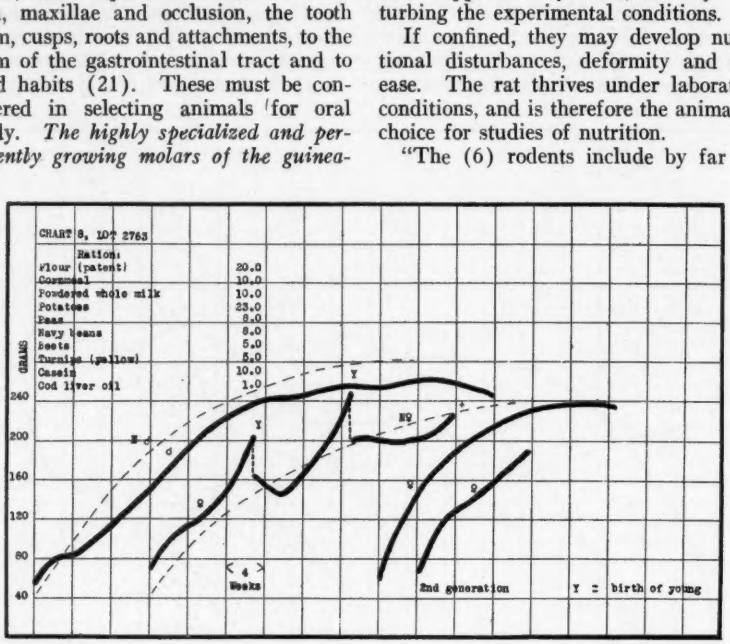

Chart 8.—Lot 2763. The diet of this group presents some very interesting features. It consisted in large part of milled cereal products, tubers and fleshy roots (potatoes) whole milk powder (Merrell Soule) a purified protein, casein, and cod-liver oil. The latter substance was included for the purpose of adding one of the vitamines (fat-soluble A). This diet was very comparable to the better class human dietaries composed of wheat flour, potatoes, meat and butter. The meat in this diet was replaced by casein. Both are essentially sources of protein and phosphorus. Growth was only fairly successful, the fertility was rather low, and the infant mortality somewhat excessive. The second generation of animals was not so vigorous as the parents.

nutritional environment, one factor of which (the rodent denture) is of advantage in obtaining and masticating food.

A (21) composite rodent dental formula consists of incisors $\frac{1}{1}$, canines $\frac{0}{0}$, premolars $\frac{0}{0}$, molars $\frac{3}{3}$, bilaterally. The canines and premolars are missing. The space occupied by these teeth in the usual mammalian denture appears as a diastema. The incisors are formed and maintained by persistent enamel organs and pulps (Fig. 1). Due to the situation of the mandible and to varying curvature, the mandibular incisors normally articulate posterior to the maxillary incisors. "The (1) glenoid cavity of the temporomandibular articulation is longitudinal rather than transverse. It follows because these teeth are constantly growing that occlusal surfaces are constantly being worn off. The elongated temporomandibular articulation is important since it allows the anteroposterior relation in gnawing. The proper regulation of length in the incisors (2.5 mm. average per week) (4) is controlled by their own interaction, a very important factor in the animal economy" (Figs. 1 and 2).

All molars of all rodents except the rat grow persistently from constantly functional enamel organs and pulps and therefore must be continually worn and renewed. They are of the lophodont or hypsodont coronal pattern, common to herbivorous animals and very unlike human teeth. In the rat the molars are completed teeth of the bunodont coronal type (Fig. 2) common to the omnivora, including man (21). They are all erupted by the thirty-fifth day (4) and are completed apically and attached by fixed alveoli by about the sixtieth day (Figs. 1 and 2). These twelve teeth

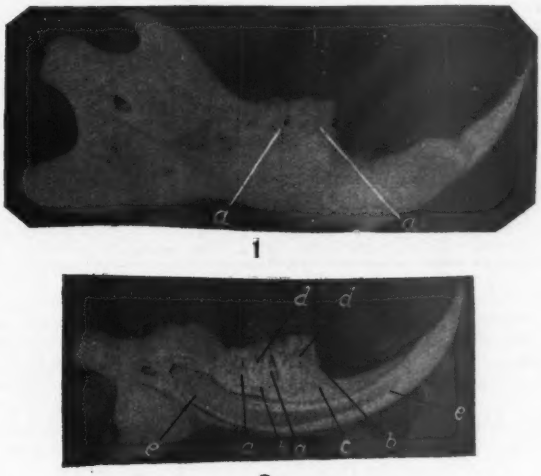

PLATE I.—NORMAL MANDIBLE OF THE RAT.

Fig. 1, the right ramus of a normal mandible of a stock rat; note the fine type bone; *a*, excellence and close approximation of the crests of attaching tissues; and various processes and angles. × 4. Fig. 2, vertical section of the left ramus; *a*, same mandible, showing the alveolar crests; *b*, width of peridental membranes; *c*, flare of the roots with pointed apices; *d*, finished pulps of molars; *e*, insertion of the incisor, with large persistently functional pulp. × 3.

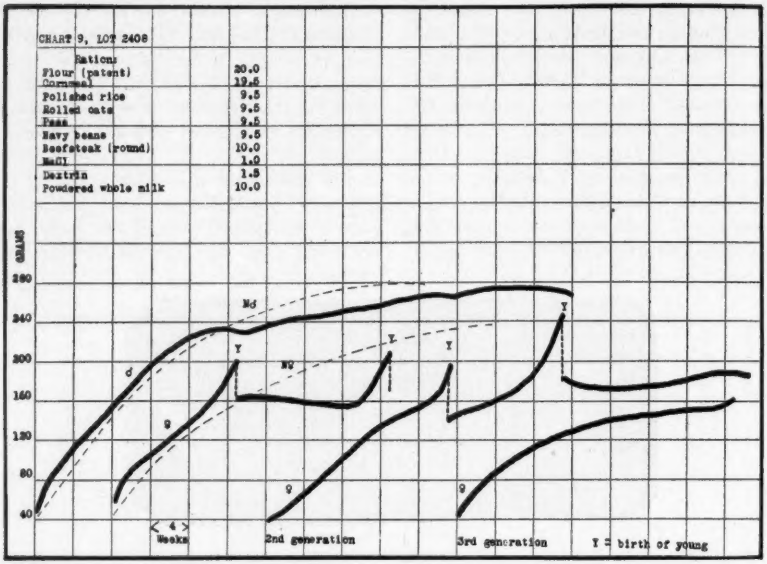

Chart 9.—Lot 2408. This diet was very comparable to such as are usually regarded as of good quality for human beings. It consisted of milled cereals, legume seeds, milk and meat. This type of diet has been extensively studied in our laboratory. It does not contain sufficient calcium or the vitamine fat-soluble A. These faults are sufficient to cause rats confined to the ration to fail to reach the maximum size or to exhibit maximum fertility. When a family is confined to such food supply thru several generations, the physical development gradually deteriorates. They show signs of senility rather early and fail to maintain a well nourished appearance. It is only necessary to introduce more calcium and more fat-soluble A into such a diet in order to entirely change the life-history of the individual and of the family restricted to it.

Such diets as are described in connection with Charts 8 and 9, and which correspond rather closely with diets common in American homes, form a very important problem from the standpoint of health and prevention of oral disease. Menus corresponding to such food mixtures look attractive and appear to satisfy the appetite. An example will illustrate this.

Broiled steak — Mashed potatoes
Buttered peas — Pickled beets — Combination salad (gelatin, peas, carrots, etc.)
Bread — Butter — Coffee
Apple pie — Cheese

Such a diet is unsuited for the developing child or for the maintenance of optimal well-being in the adult over a long period. Under such a dietary regimen early ageing and inefficiency are to be expected.

In order to correct the faults in diets of this type, liberal amounts of dairy products should be included in their various acceptable forms, as butter, cheese, milk, creamed soups, and vegetables of the leafy type in the form of greens and salads. Most important of among these are spinach, turnip and beet tops, celery leaves, romaine, collards, lettuce, cabbage, endive, etc. Since our ordinary diets are practically lacking in the antiscorbutic substance (water-soluble C) it is very desirable that some raw fresh fruits or raw vegetables enter into each day's dietary, These may best be used in the form of salads, since this affords an opportunity to introduce further leafy vegetables into the menu in small amounts.

are, therefore, comparable numerically and anatomically to the molars of man.

According to the period in which defects arise, it should be possible to state approximately whether they are developmental, or structural disturbances observed in adult teeth.

The association of two such divergent dental types in the same animal is exceptional in the mammalia. It has a high experimental value, for the effect of nutritional errors may be observed in the

about one hundred to one hundred and fifty days old. Not less than two rats of the same group was considered and many contain seven upon the same diet. All rats were closely related since our colony has been inbred without exception over a period of twelve years.

All food mixtures were finely ground to prevent selection of components. Laboratory observations and the condition of the teeth indicated that the rats had thoroly masticated the food.

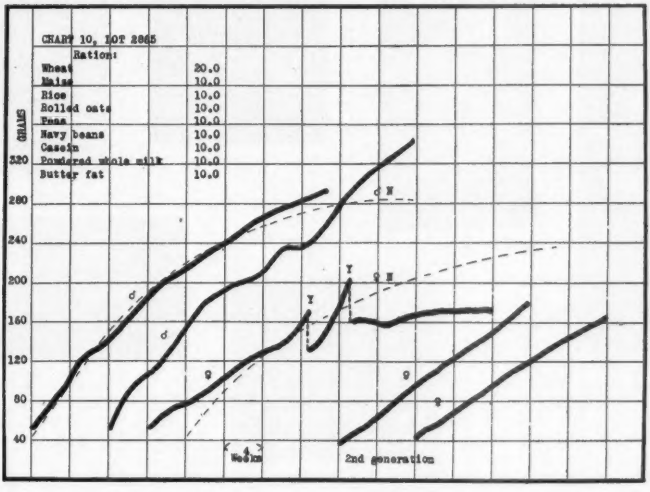

Chart 10.—Lot 2865 had a diet similar to that of Lot 2408, but whole cereals were employed instead of milled cereal products. The chief difference lay in the high content of fat-soluble A (in butter fat) in ration 2865. This improvement was of considerable significance, however. The growth was faster and the animals were in better condition than were those of Lot 2408, which had less fat-soluble A. The diet of Lot 2865 was, however, too poor in calcium, notwithstanding its content of 10 per cent of milk powder. This deficiency was serious enough to make the growth somewhat slower than the maximum rate, and the second generation failed to grow normally.

persistently growing incisors and compared with the fixed molars (Figs. 1 and 2).

Specimens were prepared by boiling or by maceration (rotting). Of the 220 rats included in these observations, about two-thirds of these were put on experimental diets at the age of forty to fifty-five days, and were continued on these diets until killed. Many adults were

Only gross defects, under the following broad headings, are reported. Molars: (1) caries-like lesions; (2) loss of attaching tissues and teeth, or both. Incisors and Molars: (3) fracture. Incisors: (4) osteodentin and pulp exposure; (5) overgrowth; (6) loss of maxillary and mandibular areas.

Percentages of defects were calculated from the number of rats and periods on

the diets and number of teeth involved. Caries-like lesions occurred only in the twelve molars, while fractures occurred in molars and four incisors, or sixteen teeth in all.

Caries-like defects. Under the heading "Caries-like Defects in Molar Group" in Table III, is shown the incidence of these defects in rats restricted to several types of defective diets discussed in the paper.

ward do not result from hypoplastic enamel. They are not absorptions by dental pulp function but have every microscopic characteristic of caries. That the defects which occurred in the teeth of these experimental rats were comparable to those found in human dental caries (Figs. 3 to 8) is seen in the minute and slowly enlarging initial lesion in any deep enamel sulcus: the rapid dentinal invasion undermining enamel

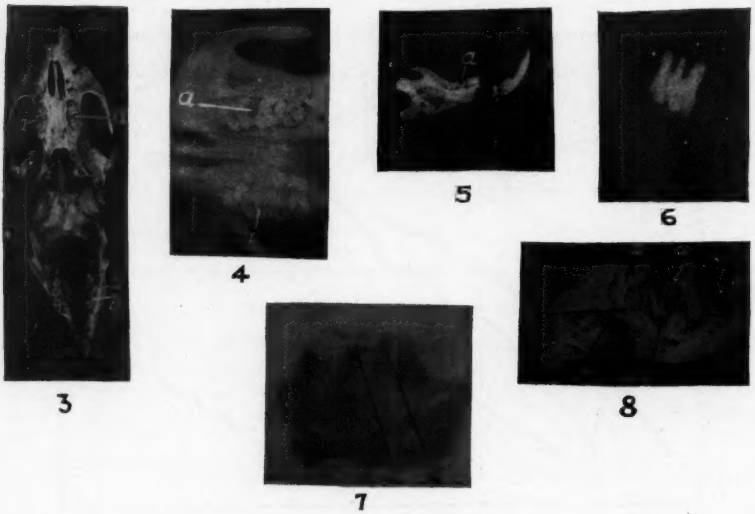

PLATE II.—CARIES-LIKE LESIONS.

Fig. 3, palatal view of the maxilla and plan view of mandible; *a, a,* distal proximal caries-like lesion first molar maxilla; *b, b,* incipient caries-like lesions mandibular molars. Slightly reduced in size. Fig. 4, palatal view maxilla: *a,* caries-like lesions proximating first and second molars; *b,* distal sulcus first molar. × 2. Fig. 5, occlusal caries-like lesion left mandibular second molar (*a*) pulp exposure. Slightly reduced. Fig. 6, left maxillary first molar, occlusal caries, broach in pulp canal. × 3. Fig. 7, lingual view of the left ramus of mandible. Fig. 8, vertical section of the right ramus Same mandible. × 2: *a, a,* deep and rapid caries-like lesions, exposing pulps. Teeth are dark and necrotic; *b,* hypercementosed apex with multiple foramina; *c, c,* open canal and apical abscesses; *e,* crests of alveolus in good contact; *d,* socket of incisor apex. This is typic pulp and apical disease. × 2.

Attention is called to the remarkably constant percentage of defects thruout the series, while no molar defects were observed in stock rats in many generations.

These lesions (Figs. 3, 4, 5, 6, 7, 8), which always proceed from without inwalls; the typic laminated carious dentinal detritus and the final pulp exposure, followed by a subapical bone involvement and alveolar abscesses (Figs. 7 and 8).

We believe that further microscopic study, now under way, will confirm these observations. Dentinal resistance pre-

venting pulp exposure appears to be of the highest order. Fractures frequently resulted from caries-like lesions, but caries rarely invaded previously fractured surfaces or roots.

The incisors even when hypoplastic or dystrophic are not involved by caries-like defects. Possibly this may be due to (1) the simple tooth form and constant use in gnawing, which prevents food debris retention; (2) to the excellent resistance maintained by persistent enamel organs and pulps. If such invasion did occur, complete repair might be expected from the same source.

The majority of lesions arise in distal sulci of the first, and central sulci, of the second molars. These sulci are much deeper than those in the third molars and caries-like defects may result from food retention. Occasionally mesial and distal surfaces are secondarily involved (Figs. 3 and 4), but initially, interproximate contact facets are not attacked as

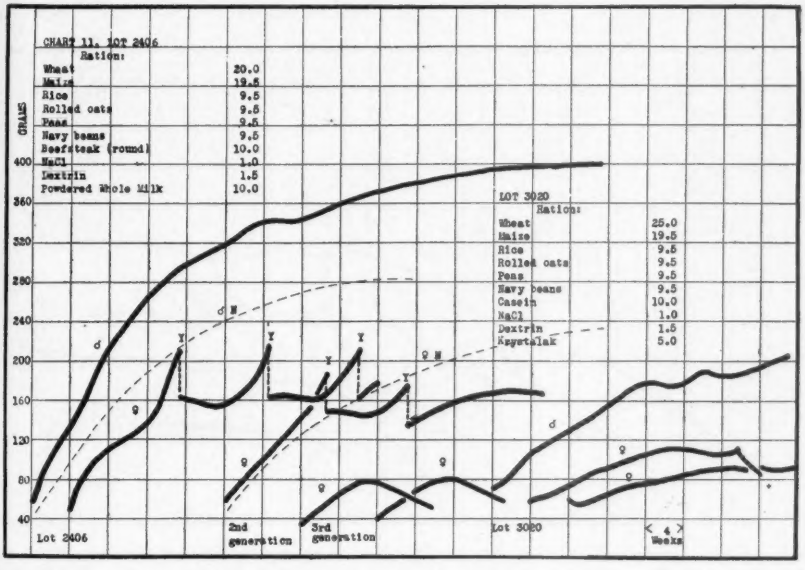

Chart 11.—Lot 2406 and 3020 represent records of two groups of rats on diets of some complexity, but in which the faults were of a twofold nature. These were insufficient calcium and fat-soluble A.

The chief source of both these dietary factors in both diets was the milk powder. In Lot 2406, 10 per cent of whole milk powder, containing butter, was included. In Lot 3020 but 5 per cent of skim milk powder was included. In other words, ration 2406 was better constituted with respect to both these factors than was ration 3020. The effect of these differences in the two diets is easily seen in the character of the growth curve and the records of reproduction.

Altho Lot 2406 grew well and were fairly fertile, the infant mortality was high and they aged early. Such young as grew up were maintained on the family diet and were inferior to their parents. They were short and stocky, and rough-coated, and their span of life was short.

Lot 3020, the diet of which was enhanced with respect to calcium by 5 per cent of skim milk powder, and had no supplementary source of fat-soluble A, was inferior. They were stunted in growth and infertile, rough-coated and aged early.

in the human. The excellence of contact maintained by attaching tissues may explain this immunity (Figs. 1 and 2).

The rat's life span of almost three years is said to be comparable to man at the age of ninety. One day, therefore, approximates thirty in the human. No doubt developmental cycles vary greatly, but rats from 120 to 150 days old may be compared with children between the tenth and thirteenth years.

The teeth of 85 per cent of all school children between these ages are said to be 25 per cent carious. The teeth of 31 per cent of this group of rats were 5.26 per cent carious; an average of twenty-four teeth in children as compared to twelve teeth in rats were exposed to caries-producing agencies. Hence the disparity between 5.26 per cent for the rat and 12.5 per cent for the child is less than it appears.

The periods during which rats are fed on defective diets and during which teeth are subject to damage are important. These vary greatly with different diets and groups, and depend upon the survival of the rat. An example is seen in the extremely rachitic and opthalmic groups, where they rarely survived over forty days, whereas on the stock diet, the animals were vigorous for seventeen months or more.

In the entire group surveyed, the longest period was 510 days for the stock, and the shortest 25 days, while the aver-

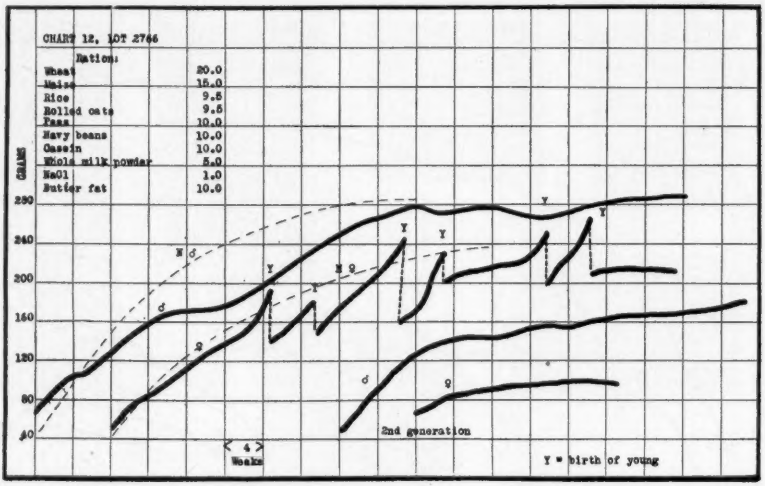

Chart 12.—Lot 2766 had a diet in which there was but one fault of any magnitude, viz., lack of sufficient calcium. It cannot be too strongly emphasized that cereals, legume seeds, tubers, fleshy roots, and meats are inadequate sources of calcium. Five per cent of whole milk in the diet did not furnish enough of this element.

The fairly good showing of these animals with respect to growth and fertility was in no small measure due to the high butter fat content of the diet. Our studies of rickets have made it clear that when the content of calcium in the diet is below the optimal or there is a serious disproportion between the calcium and the phosphorus content in the food, the presence of a high concentration of fat-soluble A tends in some measure to protect the animals against the effects of the inorganic defect.

These animals grew somewhat slower than the optimal rate. Their fertility was fairly high and the infant mortality was excessive. The few young which were weaned never appeared sleek and well nourished. Their forms were too stocky and their growth stunted.

age was 260 days. The first and second molars, those generally involved, are fully erupted about the twenty-fifth day.

All experimental rats were placed upon these diets at an average age of fifty days. It is a rapid carious lesion indeed which involves a tooth in twenty-five days (Fig. 6).

There is no reason to conclude that caries is any more rapid in the rat than in the human. On the contrary from the resistance inherent in the rodent denture, we believe it to be less rapid. The whole process is relative and can only be determined by study of the individual percentages and diets which are available.

The supposed relation of sugar to human dental caries has been postulated. It is interesting to note that cane sugar does not exist in the fifty-seven diets fed to these groups of rats (see Tables I and II).

Certain diets contained 5 or 10 per cent of powdered milk and 5 per cent of beets, but the small amount of milk and best sugar contained in these is negligible. If these lesions, after further microscopic study, show the well-known histopathology of human dental caries, it might be concluded that cane sugar was not an essential factor in these carious processes. The diets all contained an abundance of starches, which might perhaps be regarded as caries-producing factors.

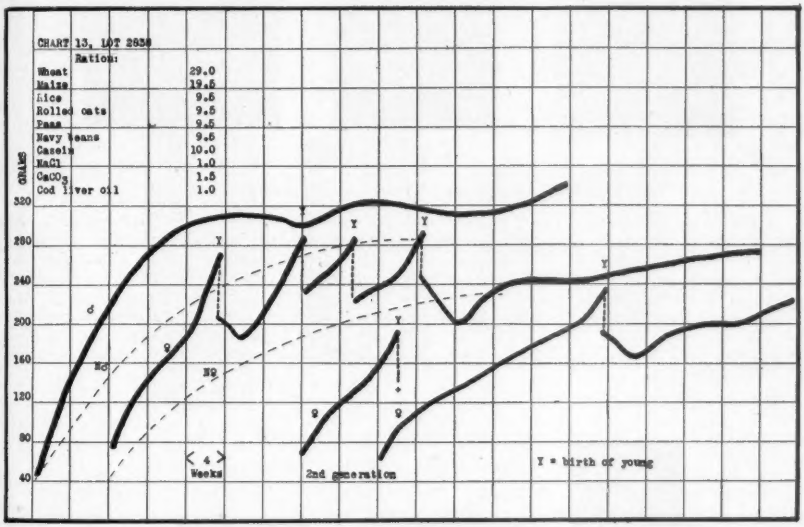

Chart 13.—Lot 2838 illustrates the effect of supplementing diets of the type discussed under Chart 11, Lot 2406, with the optimal addition of calcium in the form of carbonate. Fat-soluble A was furnished by 1 per cent of cod-liver oil. The contrast in the life-histories of the two groups of animals under discussion illustrates clearly the importance of having all factors in the diet properly constituted. In other studies from our laboratory similar contrasts are brought out as the result of making success or failure of groups of animals turn on the quality of factors other than those discussed in this paper. It may be confidently asserted, therefore, that if the diet deviates even in a slight degree from the optimal in respect to any essential factor, the results will become apparent in some degree, at some time, in the life-history of the individual.

Loss of attaching tissue and teeth, or both in molar group. Under the heading

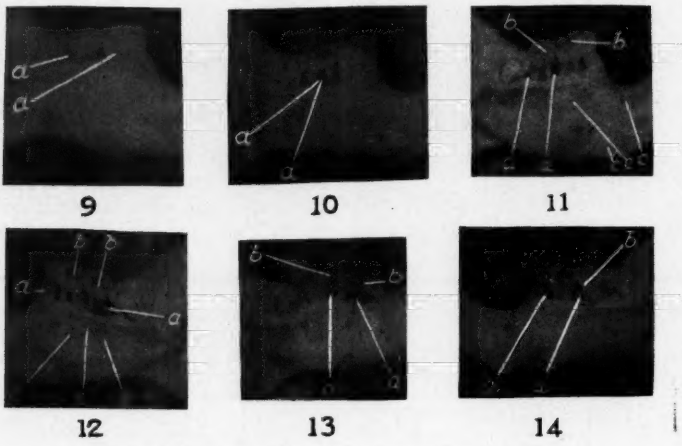

PLATE III.—ATTACHING TISSUE LESIONS ON MOLARS.

Figs. 9, 10, phases of gingival disease, atrophic type: *a, a,* loss of alveolar crests. × 3. Fig. 11, loss of all crests: *a, a,* deep interseptal invasion and loss, bucco-lingual; the exudative type; *b, b,* caries-like lesions. Note osteoporous areas (*c, c, c,*) in body of ramus. × 3. Fig. 12, typic pyorrhetic involvement with loss of crests: *a, a,* deep pockets and osteoporosity of entire ramus; *b, b,* deep caries-like lesions. Note the canalization of the cortex of the ramus (*c, c, c*). × 3. Figs. 13, 14, phases of gingival disease which may have resulted from traumatizing occlusion due to fractures (*b, b*). Pockets (*a, a*) in Fig. 13, with good type of bone suggest this factor more than in Fig. 14, where the ramus is canalized and porous and there is much loss of attaching tissues (*a, a*). × 3.

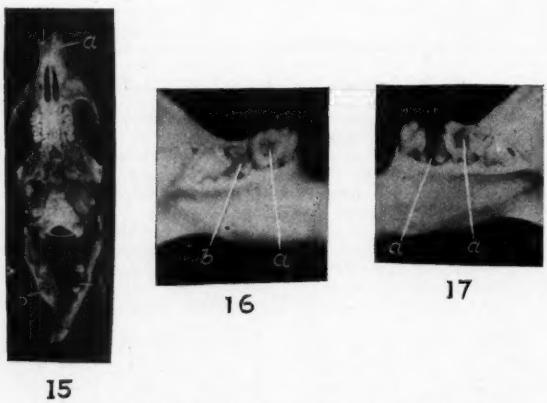

PLATE IV.—FRACTURES IN INCISORS AND MOLARS.

Fig. 15, palatal view of the maxilla and plan view of the mandible: *a,* fracture of maxillary incisor; *b, b,* fractures of mandibular molars. Slightly reduced. Figs. 16, 17, very brittle first and second mandibular molars: *a, a, a,* fractures of cusps and roots; *b,* old fractured root with much abrasion and tilting of left third molar. × 2½.

"Loss of Attaching Tissues and Teeth or Both in Molar Group" in Table III is shown the incidence of these defects in rats restricted to the several types of defective diets discussed in this paper.

Attention has been directed to the remarkable stability of the hard and soft attaching tissues of the molars in the rat. The flare of roots to meet occlusal stress is great if we may judge by occlusal facets. The stout alveolar and well-adapted gingival crests all indicate that such teeth cannot be lost without great damage to attachments (Figs. 1, 2, 8).

Osseous defects fall into two groups: (1) the atrophic type (Figs. 9, 10, 13), with progressive, horizontal and regular loss of the alveolar crests, the adjacent cortex being hard, rounded and smooth; and (2) the infective type (Figs. 11, 12, 14), with irregular, vertical invasion of the alveoli, and localized root exposure, in which the cortex is rough, soft or missing as in true exudative gingival lesions. In fact, all phases of gingival disease occur as in the human.

Age and the dietary periods must be considered in individual groups in class-

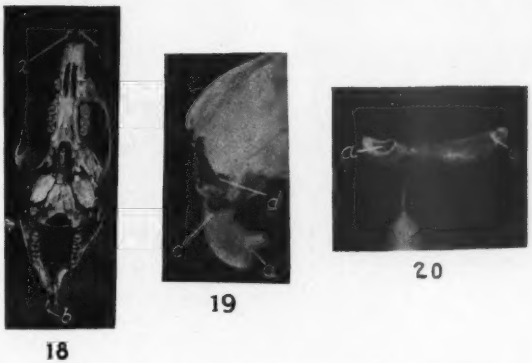

PLATE V.—PULP EXPOSURE AND OSTEODENTIN IN INCISORS.

Fig. 18, palatal view of maxilla and plan view of mandible: *a*, *a*, pulp exposure without fracture, maxillary incisors; *b*, osteodentin mandibular incisor. Slightly reduced. Fig. 19, elevation of pre-maxilla showing overgrowth of incisor, *c*, with loss of color, *a*, pulp exposure and *d*, absorption of attachments. × 4. Fig. 20, a dystrophic maxillary incisor, much shortened: *e*, hypoplastic root apex, and *a*, pulp exposure. × 3.

It cannot be said that lack of use (Fig. 9) causes attaching tissues defects; for whether the diet be soft or hard, the molar cusps of a normal rat, one year old, are abraded like those of a man fifty years.

Only such gross osseous defects as actually exposed cervical aspects of roots, or finally caused loss of teeth, are reported, and it is assumed that soft gingival tissues had been involved for some time. A microscopic study of these gingival crest lesions should be interesting and will be reported later.

ifying attaching tissue lesions. In man gingival disease generally involves the adult and thirty-two teeth must be compared with twelve molars in the rat. There are, therefore, 37.5 per cent less teeth exposed to the agencies which induce gingivitis in the rat than in man. The percentage of human gingival disease is not known but a 41 per cent involvement of the attachments of the teeth of these experimental animals, many of which were quite young, is significant.

Fractures in incisors and molars. In Table III under the heading "Fractures

in Incisors and Molars" (Figs. 15, 16, 17), is shown the incidence of this type of injury in teeth, resulting from several types of dietary faults.

A small percentage of fracture (.024) will be noted in stock rats. This is not unusual in the molars of foraging rodents. Dr. Bebb's exhibit, Northwestern University Dental Museum, illustrates these conditions in wild rats. The damage, however, is inconsiderable when compared with the percentages here reported in the groups on experimental diets. Such a high incidence of fractures in wild rats would seriously interfere with the nutrition. Altho growing persistently, there are no evidences of repair if incisors are badly fractured. Frequently these teeth are so brittle as to fly to pieces when seized by dissecting forceps or so chalky as to crumble if touched (Fig. 15). Fracture of the incisors is commonly described as existing in rodents where the endocrine glands have been removed or damaged.

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PLATE VI.—OVERGROWTH IN INCISORS.

Fig. 21, complete overgrowth maxillary incisors (*a, a*) which perforate the skull.

Many of the defects in maxillae and teeth ascribed to disturbance of endocrine function have been produced in these experimental rats solely by defective diets. *In animal experiments, all dietary factors must be perfectly evaluated before conclusions of any value as to the relation of oral disease to nutrition can be obtained.*

One of the conclusions reached by G. V. Black (2) as the result of physical tests of human teeth was that resilience and resistance to stress, rested in the organic matrix and not in the inorganic content of the dentin. That any substance so resilient as the orthodentin of the adult rat should be so modified by diet as to become chalky or brittle is remarkable. This is conceivable in the incisor group where the vascularity of the persistent pulp might modify the dentinal matrix thru the tubuli and fibrils (Fig. 2); but the causes are not so apparent in the stable pulps and fixed dentin of molars in which the greatest percentage of fracture occurs (Figs. 2, 16, 17).

Increasing brittleness of human teeth has been frequently observed as senescence advanced. Extremely hard and brittle teeth are common in all life-periods of many races, notably among the Italians (Sicilian).

A histologic study of the matrix of such dental types as compared with brittle rat molars would be valuable if the actual diets of the people were known.

Pulp exposure and osteodentin. In Table III under the heading "Pulp Exposure and Osteodentin in Incisors" (Figs. 18, 19, 20), is shown the incidence in teeth of these abnormalities in relation to faulty diets.

Pulp exposure in molars is not mentioned, for it is negligible except that which results from caries-like lesions (Figs. 5, 6, 8). Exposures which were produced by fracture have already been discussed. These were quite open and easily detected and differed materially from exposures here classified.

In normal incisors at least 4 mm. of sound orthodentin exists between the pulp apex and grinding surface (Fig. 2). If this is not normally renewed, or if it is replaced by osteodentin, such defects are significant of failure in function of the persistent pulp (Fig. 18). In these conditions, the pulp chamber is enlarged in all directions and dentinal walls are thin with consequent

shortening of the curvature of roots. The enamel is rarely hypoplastic, tho it often becomes mottled or loses color (Fig. 20). This is evidently a dentinal condition comparable to osseous disturbance, associated with enlarged medullary spaces and the formation of osteoid.

Overgrowth in incisors. In Table III under the heading of "Overgrowth in Incisors" (Fig. 19, 21, 23) is shown the

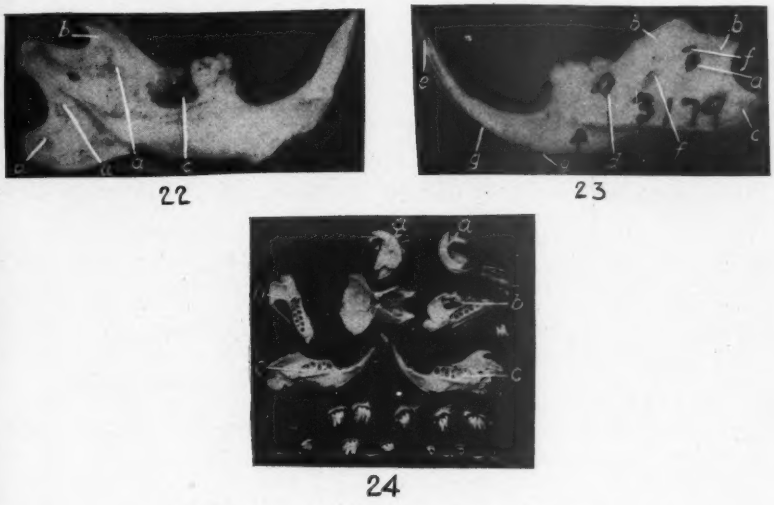

PLATE VII.—MAXILLARY MANDIBULAR DAMAGE.

Fig. 22, *a, a, a,* absorbed and osteoporous areas left lingual ramus; *b,* porosity and loss of coronoid process; *c,* old sclerosed and evidently much infected socket resulting from gingival or apical disease, second and third molars. × 2½. Fig. 23, *g, g,* loss of cortex about incisor root insertion; the entire ramus is light porous and canalized: *b, b,* absorption of the coronoid, and condyloid processes; *c,* beginning absorption of the angular process; *f,* osteoporous cortex; *a,* absorption exposing apices of molars which are normal. Area *d* should be compared with *c* in Fig. 22 which resulted from apical or gingival disease, while area *d* is maxillary disease distinct from the teeth; *e,* overgrowth of incisor. × 2½. Fig. 24, complete disintegration of an adult skull and maxilla. The premaxillae each containing an incisor, *a, a,* the palatal and alveolar processes of the maxilla, *b, b,* the porous rami of the mandible showing loss of processes and alveoli, *c, c.* The detached teeth with open apices. Slightly reduced.

incidence in teeth of this abnormality in relation to several types of faulty diets.

Overgrowth is said to result from deviation or protrusion of jaws, or loss of opposing teeth. It is also caused by pulp exposure and disease of molar attachments, both of which produce painful occlusion, and interfere with normal wear of cutting edges, which must equal incisor growth (4) which averages 2.5 mm. per week (Fig. 2).

Addison and Appleton report a series of studies on rats, confined in glass jars, presumably to prevent gnawing. The incisors were found to be as much worn by their own interaction as were those in rats eating hard food.

They note the fact that when rats were fed on bread and milk, examples of overgrowth in incisors were frequent, but when placed on varied diets, overgrowth rarely occurred. Bread and milk, may or may not be a good diet dependent upon the character of the bread and milk. This reported overgrowth may result from defective diet rather than soft food.

An elongation of the incisors sufficient to prevent contact of grinding surfaces in the molars (Figs. 19, 23), which do not grow persistently, may induce malnutrition if not starvation in the rat. In the stock rats, the elongation was not over 2 mm., while in the experimental rats the maxillary incisors were frequently so overgrown that the recurved cutting edges perforated the skull (Fig. 21).

Obviously conclusions as to the effect of the diet are useless if starvation is induced in the rat by defective teeth.

Maxillary and mandibular damage. In Table III under the heading of "Maxillary and Mandibular Damage" (Figs. 22, 23, 24) is shown the incidence of lesions of these types and their relation to several types of faulty diets.

The histopathology of these defects will probably be found to vary from osteoporotic and osteosclerotic states thru the phases of bone pathology similar to those we have described in studies of rickets and related conditions.

The cortex of the mandible and maxilla was thick, roughened, distorted or sclerotic with much repair, or it was canaliculated and porous or had completely disappeared. The medulla was much enlarged and frequently the entire bone was spongy and light with many trabeculae. The affected areas were remarkably symmetrical and bilateral (Fig. 22).

In the mandible, the cortex of each ramus was frequently missing along the incisor root insertions and root apices were exposed on the external oblique lines (Fig. 23).

In extreme cases resorption of the coronoid, condyloid and angular processes occurred, and the rami were frequently so softened, that it was questionable whether the mandible could have functioned in mastication (Fig. 23).

In the maxilla, there was loss of the premaxillary buccal plate, exposing incisor root insertions. Median separation of the premaxillae and palatal processes occurred and absorption of the palate bones in the floor of the nares was not uncommon (Fig. 24).

In both mandible and maxilla, bilateral areas of resorption appeared lingually and buccally in the body of the bones, which could not have been caused by dental disease. These must be sharply differentiated from enlarged sockets and tracts of previous fistulae in the subalveolar bone induced by gingival disease or alveolar abscess (Figs. 22, 23).

The defective maxillae of these rats should be compared by radiographic study with the maxillae of children suffering from lack of development of the jaws, maxillary and mandibular protrusion and malocclusion, for it is likely that these osseous defects may be correlated.

It is next to impossible for the orthodontist to stimulate bone growth or establish normal occlusion in the jaws of these children. The teeth may be placed in normal relation by appliances, but as soon as these are removed, the entire denture drifts back into malocclusion (Fig. 22).

In adult human maxillae, rarefied areas surrounding the apices of well-filled pulpless teeth are discovered by accurate radiography. These are interpreted as alveolar abscesses and oral infective foci, and the teeth removed as a possible cause of focal disease. The contents of many such areas are frequently found to be sterile by the most careful bacteriological technic (Fig. 8).

It is possible that these rarefactions (Fig. 23), which also are often located in other parts of the maxillae than those adjacent to the teeth, may arise from dietary deficiencies and defects in man as they apparently result in these experimental rats.

In Table IV under the heading "Total Damage—Averaged from the Six Groups of Defects, and Fifty-seven Diets," is shown the incidence of damage in relation to specific types of faulty diets.

The bone ash of the hind-leg, pelvis,

TABLE IV

PERCENTAGES OF ANIMALS EXAMINED, WHICH SUFFERED FROM SPECIFIED DENTAL DEFECTS
(DATA BASED ON OBSERVATIONS ON 220 RATS)

Caries like defects in molar group	31.0
Loss of attaching tissues or teeth or both in molar group	41.8
Fractures in incisors and molars	29.5
Pulp exposure and osteodentin in incisors	22.7
Overgrowth in incisors	20.0
Maxillary and mandibular damage	25.0
Total damage averaged from the six groups of defects:	
Males	79.2
Females	84.1

scapula, and mandible of groups of rats on each of the fifty-seven diets was carefully determined. In stock animals with good bones and teeth, the ash content averaged 66 per cent for hind-leg; 62 per cent for pelvis; 65 per cent for the scapula and 74 per cent for the mandible. In the rats on deficient diets, these percentages were from 5 to 15 per cent less for all bones, save the mandible, which was constantly from 6 to 8 per cent higher, presumably because of the presence of teeth.

With the exception of the group of diets, in which the deficiencies were protein of poor quality and too little fat-soluble A, the fifty-seven diets reported in these experiments were used by McCollum and Simmonds and Shipley and Park (15, 16, 18, 19, 20) in the study of rickets and related osseous pathological conditions.

As the oral defects herewith reported occurred in the same groups of rats, a comparative summary of the findings in all their reports follows:

These reports have shown that the internal structure of the skeleton of the rat can be changed at will by varying the ration which the animals received. In other words, bone is an extremely labile tissue and is readily influenced by nutritional environment.

Some of the faulty diets which were studied produced rickets often of an exaggerated type, others caused osteoporosis. Still others resulted in the development of the peculiar lesion, which has been called osteosclerosis.

A study of the effects of these diets on the skeleton would indicate that the growth of the skeleton was dependent on at least three substances (15): (1) an organic substance present in certain fats which may or may not be identical with the anti-exerophthalmic, fat-soluble A, (2) calcium, and (3) phosphorus (18).

If this organic factor is low in, or missing from, a diet, the structure of the osseous tissue is dependent on the ratio between the above-mentioned ions in the diet and the circulating blood.

If calcium is present in amounts equal to, or exceeding those which would be optional for growth and function (16), if all other factors are satisfactory but the phosphate ion is low, rickets is produced (21).

The same disease results when the converse relation exists between the two ions (20).

In other words, diets which contain optimal or excessive amounts of calcium but are low in phosphorus and the organic factor, produce rickets.

Rickets also results from feeding diets low in calcium and the organic factor if phosphorus is present in satisfactory amounts.

Diets which are satisfactory except that they are deficient in the organic substance result in osteoporotic but perfectly calcified bone.

Diets which have a comparative deficiency in calcium but are very high in phosphorus and in the organic substance produce so-called osteosclerosis, with large numbers of small imperfectly calcified trabeculae (19).

Diets deficient in calcium alone pro-

duce a pseudo-rachitic condition with overproduction of osteoid.

The studies of diets which contain varying percentages of calcium and calcium phosphate show that the absolute amount of either ion in the diet is of relatively little importance as compared to the ratio which exists between the two. That is, normally calcified bone is produced without regard to the reduction or diminution of either calcium or phosphate in the food, provided the content of the other ions is proportionately augmented or depressed.

When these findings in the bones are compared with the average damage on all diets, Table IV, we note the following facts:

Conclusion

The percentage of oral defects was greatest in those rats fed the diets included under the first heading, i.e. those deficient in protein, calcium and fat-soluble A. The rats on diets low in calcium exhibited the next highest incidence, and those on diets low in both calcium and fat-soluble A had the third greatest percentage of damage.

The diets low in calcium and high in fat-soluble A: those containing low or defective protein, together with low fat-soluble A, those in which the only deficiency was low fat-soluble A, form a middle group producing 11.1 per cent oral defects. The oral tissues were the least damaged by the diets high in calcium and low in fat-soluble A; those high both in calcium and cod-liver oil and those low in calcium and in cod-liver oil.

No caries-like lesions, pulp exposure or osteodentin, attaching tissue or maxillary defects occurred in our stock rats. The 0.36 per cent of defects in stock rats resulted from fracture or overgrowth.

The relation of the elements, calcium and phosphorus to each other and of both to fat-soluble A, evidently has a most important bearing on dental development and maintenance.

This is beautifully illustrated in the remaining group of eighty stock rats of the 300, who were variously placed on diets in which fat-soluble A, in form of butter fat or cod-liver oil, was replaced by like percentages of animal and vegetable oils. When olive, cotton seed, and peanut oils were used, percentages of oral defects were high, particularly with peanut oil. With burbot, or shark liver (fish) oils, they were much lower, altho none of these maintained dentures equal to the stock rat.

Very similar results were obtained by lowering calcium percentages and substituting salts of magnesium and strontium. Magnesium disturbed the bones profoundly, with little dental effect, while strontium substitution did not involve the maxillae or denture, but when not more than 2.5 per cent calcium phosphate replaced 1.5 per cent calcium carbonate in diets which barely approached the plane of the minimum physiological requirement, oral results were the most disastrous and the most pronounced found.

The idea must not obtain that diets which produce bad bone produce interstitially bad teeth, for the reverse is equally true and the question is not settled.

The relation of the antineuritic and antiscorbutic substances to oral disease cannot be discussed in this report. A deficiency of the antiscorbutic substance from the diet of man would no doubt be a factor in the production of oral disease, but the rat, as has been noted, is able to synthesize this substance.

It is our belief, however, that severe oral disease may result from diets which are only relatively defective, where the disturbance appears to be out of all proportion to the cause.

In these borderline phases, the *dietary defect or deficiency is minute and can only be determined by careful scrutiny of the diet and the patient, or animal, over a considerable period.*

It is not possible at this time to name any one deficiency which specifically causes dental or oral disease; it would

appear that *any slight variation in the American diet, which always so dangerously approaches the level of dietary deficiency, might become active at any period of lowered resistance or of physical or nervous stress.*

The present paper forms a preliminary communication of some of the results thus far obtained. We have not as yet made a detailed analysis of the relative importance of each of the essential factors of the diet in the formation of the teeth, but enough has been learned to *point clearly to the diet as the agency of primary importance in determining the quality of the teeth and their vitality, as shown by the effectiveness of their barriers against invasion by micro-organisms.*

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SOME IMPORTANT FACTORS WHICH INFLUENCE OCCLUSION

By G. S. MONSON, D.D.S., St. Paul, Minnesota

(Read before the National Dental Association, Milwaukee, Wisconsin, August 15-19, 1921)

WHENEVER we find a man confining his efforts wholly or in part to full denture construction, we are bound to find a certain amount of grief and disappointment.

We have men working to perfect various technical details of our work, such as impressions, aesthetics, etc. As we perfect ourselves technically we gradually eliminate much of the grief just mentioned.

My research has convinced me that with these many details of technic must be combined reconstruction, keeping in mind anatomical relationships and physiological function.

Reconstruction on this basis with full dentures is bound to bring grief and disappointment in proportion to the operator's lack of knowledge of the anatomy and physiology of the parts involved. This knowledge must include the framework or osseous structure, origin and insertion of the major and depressor muscles of mastication, the structures of the throat and neck both superficial and deep.

Physiologically, we must know what is the normal action of the tributary organs to the mechanism of mastication. This includes the salivary glands, the tonsils, the tongue in the act of swallowing, the drainage, by swallowing, of the mouth, the posterior nares, and the Eustachian tubes. Finally, what makes the maximum efficiency of the air passages?

Our study of anatomy and physiology is with our subject in perfect health and no deformities. We are *then* ready to look for and recognize conditions which are evidence of pathology or at least deformities.

Let me at this time emphasize an important point with this question. "How can any man expect to prepare his patient mentally and carry her over the period of return to normal, without a thoro knowledge of the anatomy, physiology and pathology involved?"

It is only after careful study that the operator can reconstruct the lost facial dimension, which is merely the facial evidence of change in anatomical relationship. This in turn impairs functional action of the total organism, instead of as is commonly considered merely an impairment of the act of mastication. We are prone to accept as a test of efficiency for our dentures the lack of sore spots and the fact that our patient is satisfied with her efforts at mastication. We forget that our dentures should be a restorative prop as near like the original was when at its best as we can make it.

This restoration of facial dimension very often brings criticism from our patient, her family and friends, unless we

educate them *all* by educating the patient to what we are trying to accomplish. *We* must know and make our patient understand that the change in the face which they criticize is but temporary.

When we use the term *restore* we are endeavoring to as nearly as possible place the mandible in its normal relation with the rest of the osseous structure of the face and cranium. This permits in a comparatively short time, depending on the amount of dimension lost, the integument and superficial structures of the face to assume the original position they were in before facial dimension was lost.

It is a fault with a great many operators that rather than go thru the trouble of educating the patient to wear dentures constructed with real restoration in mind, they will compromise for present appearance, many times making no restoration of lost dimension whatever. It is better with a patient whose mental attitude is hard to influence, to construct a first set of dentures making a slight restoration until the patient can be won over; a series of dentures then with a little more restoration each time will in the end give the result desired.

In studying the subject of the construction of the human mechanism of mastication, it is a question whether to approach by a study of the bones forming the framework, or the study of the muscles which played such an important part in the development of those bones and all parts adjacent. A little thought will convince one that during the age of development, exercise of muscles creating stress on their origin and insertion must of necessity develop the bone to meet this stress. Even after the period of development the bones may be modified in form and relation due to nature's effort to compensate for abnormal stress, caused by muscular unbalance. This is evident when the relation of mandible to the base of the cranium and facial bones has been changed from its original by loss of teeth or improperly constructed dentures. Always bear in mind that the masticating apparatus was developed to its point of efficiency with natural teeth. Part or total loss of these natural teeth produces a condition which, if allowed to exist will not only give a change of relationship of mandible to the maxilla, but also a modification of the form of the bones. Further, the functions of mastication, deglutition, and respiration are all impaired in proportion to the amount of this changed anatomical relationship.

In discussing osseous anatomical relationship as regards the masticating apparatus, we must bear in mind that the only actual contact is in the glenoid fossae. A normal position in this relationship does not permit encroachment of the condyles on the external auditory meatus. This normal position is maintained by normal occlusion, because the only other contact of the mandible with the bones of the cranium is thru the occlusion of the teeth. It is evident then that any change in occlusion due to wear on or loss of natural teeth or improperly constructed dentures will permit a change in the position of the condyles in the glenoid fossae. In thinking of occlusion, we are apt to confine our thoughts to the ability of the mandible to close up to the point of teeth in contact, giving little consideration to a tooth length which will restore the normal relationship of mandible to cranial and facial bones. In other words, occlusion is more than the ability of placing the teeth in contact in the act of mastication. A further result of normal relation of mandible to the rest of the osseous framework is the normal position of all structures of the face and neck which depend for this position upon the mandible.

In Figure 1 a skull was used with the

view of the underside. Upon this has been erected a base of Bonwill triangle. Geometrically, we have determined the center of our base triangle. From the mesio-incisal angle, the center of the triangle and the line passing thru the axis of the condyles, we project lines to similar points on the same skull in lateral view. The value of this diagrammatic figure in our present discussion comes from our projected center of applied force. A little study will convince one that with such a center to figure from it is easily demonstrated how great is changed osseous relationship because of shortened occlusion. This, of course, would be evidenced by a shortening of the line between the mesio-incisal angle and the center of applied force.

With this in mind, we arrive at the deduction that with this shortening of occlusion, producing a shorter line from mesio-incisal angle to center of applied force, we also will have a shorter distance between the origin and insertion of many of the major muscles of mastication.

Anatomy teaches that the term "origin of masticating muscle" is meant to imply its more fixed or central attachment, and the term "insertion," the movable point to which the force of the muscle is directed. This is important because we should have a clear mental picture of muscular anatomy in order to thoroly understand change of muscle tone which leads to unbalanced muscle energy.

Considering change of position or relation of the mandible, we have two principal groups of muscles to study (Fig. 2). The great fan-shaped temporal radiating from its insertion in the coronoid process to its large spread out origin in the temporal bone, porduces a pull upward and backward. Opposed to this, we have the group composed of the internal and external pterygoids and masseter. This group produces an action which is mainly upward and forward. These two forces, when all muscles involved are of equal or normal tone, gives what we term muscular balance controlling the main movements of the mandible.

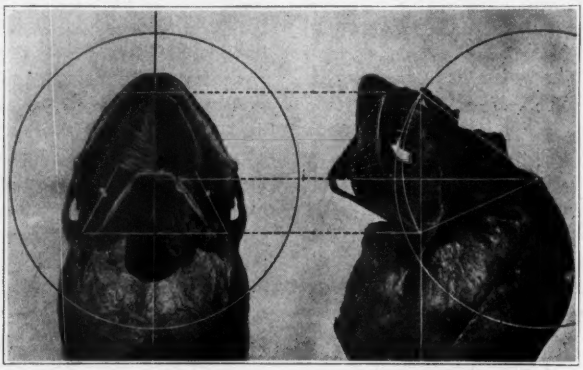


Fig. 1

Figure 3 shows the external pterygoid with its horizontal position from origin in the sphenoid to insertion in front of

the neck of the condyle, producng the forward movement of the mandible as in excising. The internal pterygoid with greater part of the ramus having a main action to close the jaw but with considerable force directed forward. This last

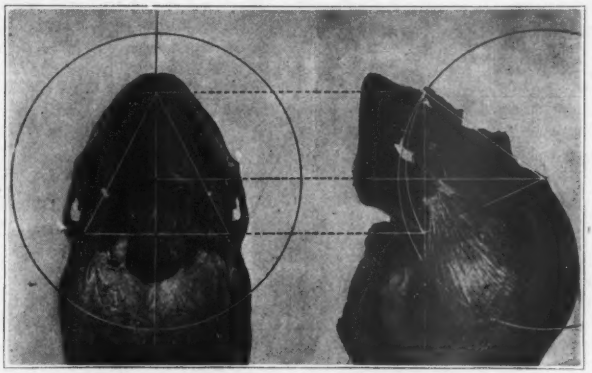


Fig. 2

its direction from insertion in the lower and back border of the angle of the mandible to its origin in the pterygoid fossae of the sphenoid also near the tuberosity group we are most concerned with in our study of loss of muscle tone due to lost facial dimension. Any change of position causing the mandible to occupy a

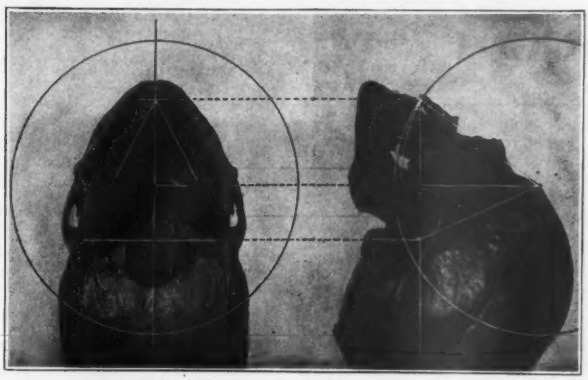


Fig. 3

of the superior maxillary produces a pull upward and forward. Figure 4 is the masseter, with its origin in the zygoma and malar process and insertion in the closer relationship to the maxilla than normal will naturally shorten the distance between the origin and insertion of the masseter and internal pterygoid. If

we liken a muscle in its normal position as to its tone or muscle pull to a rubber band stretched to a certain position, we can readily see how the muscle will lose tone by lessening the distance between points, the same as the rubber band would by lessening the distance between the stretched ends. This loss of normal tone of these two muscles allows the horizontal fibers of the temporal to overbalance, and we have the first step in the backward movement of the mandible. As lost facial dimension progresses the temporal gaining in its overbalance, we get a shortening of distance between origin and insertion of external pterygoid as well, which condition when pronounced completes our vicious circle of deformity.

Conversely by opening the relationship of mandible to maxilla using as guide relief of condyle encroachment, we gradually put normal stretch back on these muscles. Soon muscular balance returns between groups and the mandible takes its normal position with condyles in their normal position in the glenoid fossae.

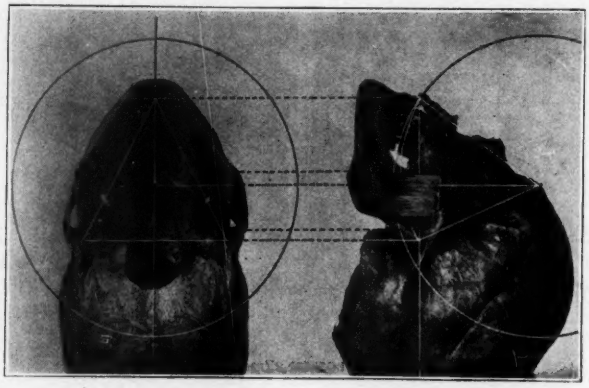


Fig. 4

Discussion

Russel W. Tench, New York City: The very vital suggestion made by Dr. Monson that the prosthetist should have a thoro foundation in the anatomy of the structures that affect our work will bear emphasizing. It is essential that we have a clear mental picture of the origin, insertion, and action of the muscles that influence the success of our efforts, and also of the mechanics involved in the action of these muscles individually and in groups. In addition to this, we should have a thoro knowledge of the effect of applied force on the stability of dentures, as modified either by the shape or the relation of the opposing ridges.

Dr. Monson advises an opening of the bite which is greater than has commonly been accepted as desirable. As a guide, he suggests the intrusion of the condyle upon the auditory canal. To bring out exactly what his technic is in determining this, I would like to know whether he studies the intrusion at the time the teeth are in contact, or when there is a clearance between the upper and the lower sets of teeth, as during speech, or when the mandible is at rest, with the face in repose.

To make myself clear, each of you without changing his vertical facial dimension at all, if his face is in repose, can slide his tongue between the occlusal surfaces of the bicuspids and molars. When your face is in normal repose, your teeth are not in contact. Under these conditions, there is little or no tendency of the condyle to intrude upon the auditory canal in a few cases that I have examined.

The statement is made in the paper that the loss of facial dimension impairs the function of the total organism, meaning, I presume, the health of the patient. The question may arise, "In what way does this come about?" and if it

does, how are we to recognize the systemic symptoms produced by such a loss of facial dimension? I believe that if this can be made clear, it will assist us in seeing and discharging our duty to our patients in this respect, for it is certain that both tact and perseverance may be required on the dentist's part in many instances, if the bite must be opened more than heretofore considered right.

Dr. Monson says: "We forget that our dentures should be a restorative prop as near like the original, when at its best, as we can make them." I presume in making this statement, Dr. Monson refers to normal cases, and not to cases in which, for aesthetic reasons, it is desirable to resort to surgery to *reduce* the bite opening.

Another point that I would appreciate further enlightenment upon is how the functions of mastication, deglutition, and respiration are impaired by changed anatomical relationship of the mandible to the maxilla. I do not know that they are materially impaired, but if they are, I want to know how.

These queries are put to gain information, not in the spirit of criticism. I am interested in developing my own technic, and to that end do not wish to let slip by this opportunity to have these points made clear.

Dr. Monson (closing): The first question was in regard to the normal relation in opening the bite. I contend that the normal position in a normal mouth, under good development is in occlusion. In opening these dentures and opening the mandible in relation to the maxilla there is tension, there is atrophy of those muscles, and we are not going to have any restoration until they are put back to their normal relation. There is always enough elasticity for movement of the muscle to give us a rest position, and in all development cases that I have had a chance to observe the natural jaws, the best development of the cranium and the mandibles were in relation to rest position. Interlocking of the cusps carries on the harmony of development of the two bones.

With regard to the functions that are impaired, let me cite an extreme case in my practice. A lady was sent to me by a nerve specialist. She could neither sleep nor rest. There was a roaring or slight buzzing in her ears. She was about thirty years of age. I opened the mandible in that case until I felt the impulse on the meatus, that is the anterior wall of the canal; it was three-eighths of an inch restoration. It was a painful experience to listen to her swallow. The hearing was improved immediately on putting in the dentures, by lifting the stress off the anterior wall of the canal, from nine inches to eighteen inches on one side, and from eleven inches to twenty-four inches on the other side. In a very short time she was swallowing like any normal individual; and there was a gain of twenty-four pounds; there was a better appearance, which is only a facial evidence of the mandible in its proper relation. In a short time there will probably be a space; in the event that she does not use those muscles and they lose their tone, there will be an inaction or dropping away of that occlusion.

DIAGNOSIS OF LATENT INFECTION ABOUT THE JAWS: II[1]

By SIR KENNETH GOADBY, K.B.E., D.P.H., Cantab., M.R.C.S., L.R.C.P., London, England

(Read before the National Dental Association, Boston, Massachusetts, August 23-27, 1920)

THE first radiographs of infected bone showed that, associated with the infective process, rarefaction of bone was a striking feature. Even in simple streptococcal infections this loss of opacity to X-rays (in some instances, at any rate, due to loss of lime salt) may be excessive, but does not always indicate destruction of bone nor that it is destroyed and incapable of regeneration. This is well illustrated by this septic thumb—a common streptococcal infection, showing commencing translucency (Fig. 28), almost complete lime salt loss (Fig. 29); returning re-deposit of lime salt following healing (Fig. 30).

Radiographs of jaws affected with chronic streptococcal infections of mixed type anaerobic bacteria such as B. fusiformis and B. necrosis commonly show areas of translucency, but great care must be exercised in assuming that every such area is an actual cavity.

Figure 31 shows certain translucent areas, as does Figure 32. On removing the molar in Figure 31 no cavity was found, the bone was excessively hard and required cutting with mallet and chisel, and there was no cavity leading to the inferior dental canal. I have no doubt that most of the apparent cavities in Figure 32 are similar in nature and are possibly due to the action of certain materials used for root filling producing a chemico-physical change. Clinically, there is no tenderness, no periostitis, no loosening and, more important still, none of the blood changes to which I shall refer later. Increased translucency due, in all probability to loss of calcium salts, may follow tooth extraction, especially with pre-existing infection.

Figure 33 shows loss of opacity following the removal of a split root. There was no special difficulty in the operation; three weeks later the wound was unhealed, the radiograph showing progressive extension of rarefaction. The irregular appearance of the outline is characteristic and should be compared with the regular outline of the translucent patch in Figure 31.

Sclerosis, or increased density of bone surrounding latent jaw infection, is indicative of repair—the balance of equilibrium is in favor of the tissues. Figures 34 and 35 show various degrees of sclerosis; in Figure 34 the thickening is pronounced over the root of the first molar; Figure 35 exhibits sclerosis and porosis. These two cases were treated without extraction with the exception of the first molar in Figure 37. All three patients are at the present time (six years have elapsed), in normal health, there is no pus in the jaw, and the symptoms of periarticular arthritis, toxemia and anemia, have entirely disap-

[1]Part I was published in *Jour. Nat. Dental Assoc.*, May, 1922, p. 371.

Fig. 28

Fig. 30

Radiographs of Latent Infection in the Jaw
(Radiographs by S. Archibald Reed, R.B.E.)

peared. Such cases are representative of a very large class.

Before leaving the radiographic diagnosis of latent jaw infection I wish to

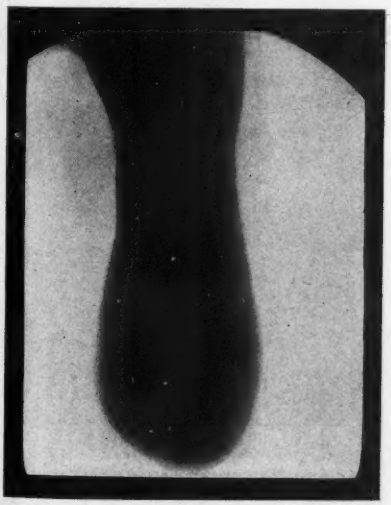


Fig. 29

Female at 26

Fig. 31.—Slight osteoporosis with some sclerosis translucent areas second premolars and anterior root first molar.

Female at 20

Fig. 32.—Early vitamine deficiency all the teeth well erupted without enamel and deficient abnormal dentin. All the incisors and premolars have been devitalized and crowned. There is a sinus pointing between the canine and first premolar. There is no loosening of any of the teeth.

Female at 30

Fig. 34.—No clinical symptoms of pyorrhea, no pus in alveolar process. Sub-acute periarticular, polyarticular arthritis. Note sclerosis around first lower molar as well as alveolar wasting.

Female at 26

Fig. 33.—Radiograph taken two weeks after removal of second premolar (crowned) simple extraction for split root. Chronic alveolar infection. Note extension of bone porosis following extraction. Periarticular arthritis.

Female at 28

Fig 35.—Localizes alveolar porosis with sclerosis symptoms of chronic toxemia and myocarditis.

refer briefly to one other type of bone porosis. Not a few cases of destructive lesions of the optic nerve have been traced to jaw infection; I have notes of ten such cases but will only quote two These two cases are both females over sixty years of age; the eye changes are retinal degeneration of destructive type. The first case had a history of pyorrhea and general extraction twenty years previously. The jaws have undergone

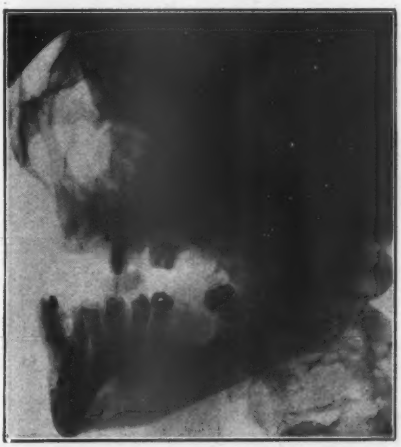
Radiograph of Edentulous Jaw—Muscle Wasting

Female at 65

Fig. 36.—History of severe pyorrhea alveolaris at 40 and all teeth extracted. Recent failing eye sight commencing at 59—diagnosis optic nerve degeneration.

progressive atrophy; there was a history—so common in the edentulous pyorrhea jaws—of recurrent attacks of sore mouth, progressive alveolar shrinkage and many new dentures. The radiograph (Fig. 36) shows the bone changes, which I have no doubt are a slow, progressive change following infection, and contributed to by disease, an exactly parallel process to that taking place in infected bone in war wounds. The second case was not edentulous; there was active suppuration which could not be described as latent; pus was present around the upper molar root (Fig. 37). Radiographs of these two cases have a common factor—the extreme thinning, in one complete disappearance of the anterior and posterior clinoid processes in the pituitary fossa.

Figure 38 is a normal pituitary fossa; Figure 39 is the pituitary region



Female at 61.

Fig. 37.—Chronic rarefaction of jaw around root of first molar (left side). Other evidences of chronic infection are seen in the lower jaw. History of chronic nasal discharge many years, 30 at least. 59 commencing optic atrophy diagnosed as inflammatory. Left eye nearly sightless, partial vision on right eye.

of the first case, Figure 40 that of the second. The association appears significant in view of the anatomical relation of the optic chiasma to the pituitary fossa, and the frequent appearance of osteoporosis in jaw infections.

In the diagnosis of suspected latent jaw infections a radiograph is essential; films alone are misleading. The radiograph should be stereoscopic and allow a view of the jaw as an anatomical and physiological whole.

Perhaps the most widely canvassed disease attributed to jaw infection is

Fig. 38.—Radiograph of normal skull showing well defined pituitary fossa. Compare with Figures 39 and 40.

arthritis. A suggestion put forward in my Hunterian lecture (7) that bacteria

Fig. 39.—Radiograph of skull showing porosis of walls of pituitary fossa, the clinoid processes are just visible. Compare with normal pituitary fossa Figure 38.

living on periarticular tissues such as tooth sockets became specially adapted to infect periarticular structures in other parts of the body has been recently further developed by Rosenow (20) and others (15). Whether this is a fact, as I believe, or not, arthritic changes, atrophic and hypertrophic, end-articular and periarticular, may certainly be traced to mouth infections in a number of instances, and I shall later return to

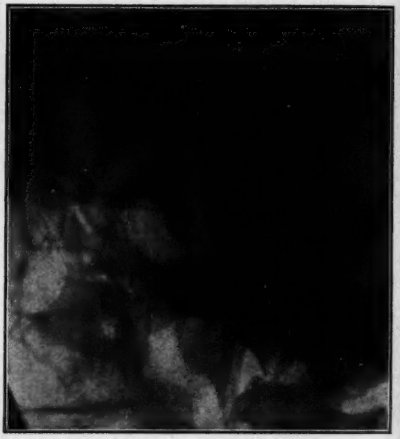


Fig. 40.—Radiograph showing porosis of pituitary fossa of last Figure. Compare with normal pituitary fossa Figure 38.

this point, but in passing would again insist on the osteoporosis of the local septic focus, so well seen in infected wounds and septic jaws. Hypertrophic affections of the alveolar process are rare, and I have only met with four cases with serious hypertrophy of the alveolar margins comparable to osteophytic outgrowths of, say, the hip joint. The great majority of polyarticular arthritis occurring in pyorrhea alveolaris is primarily periarticular with osteoporosis; thus Figures 41 and 42 are two elbow joints, both females; in

both instances the affection was traced to streptococcal infection of the jaw.

Fig. 41.—Radiograph of right elbow joint, polyarticular arthritis pyorrhea fibro-anchylosis, Female at 35.

Figure 43, the right hand, and Figure 44, the right foot of another case, are of streptococcal origin. There was no pyorrhea nor latent jaw infection, and as I found a severe bowel infection, the needless but contemplated extraction of the teeth was obviated.

Figure 45 is an experimental arthritis in rabbits one and one-half or two and one-half years after infection with a mouth streptococcus; hypertrophic as well as atrophic changes are present.

Fig. 42.—Radiograph of left elbow joint, polyarticular arthritis pyorrhea fibro-anchylosis not quite complete—porosis.

Fig. 43.—Radiograph of right hand.

My own experience, and that of practically all those who have investigated jaw infections, coincides in ascribing the chief role to the streptococcus. The term "streptococcus," however, must be used only to connote a morphological type, and much labor has been expended on the streptococcal group as a whole toward separating it into subsidiary groups. When going into the vexed question as to proper grouping, certain broad facts may be assumed, upon which most bacteriologists are, at the present time, agreed.

Firstly, the grouping into hemolytic and non-hemolytic streptococci, first suggested by Schott Muller (18), is generally accepted as a preliminary division. The work of Gordon (8), Andrews and Horder (2) in England, and later, Rosenow (21), Holman (12), and Henrici (11) in America, has established that sugar fermentation tests may be used for the differentiation of the groups of streptococci. Further, Havens

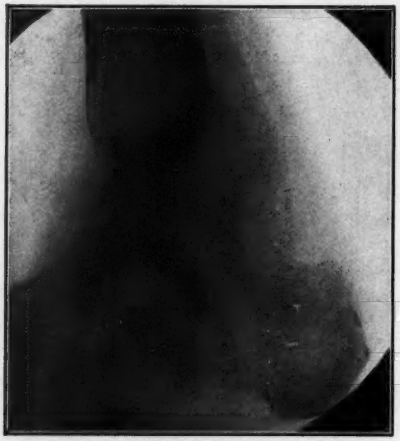


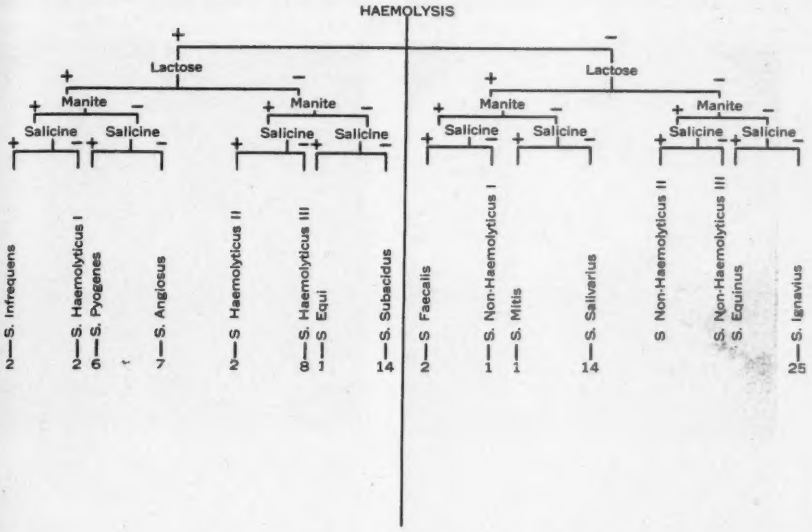
Fig. 44.—Radiograph of ankle joint.

Fig. 45.—Experimental arthritis, rabbit, one and a half years after inoculation with mouth streptococcus bacillary form.

(9) tested 292 strains of hemolytic streptococci obtained from numerous sources and grouped them into four categories with specific serum reactions. Many other observers also agree that hemolytic streptococci show far greater similarity in their agglutinative reactions than do the non-hemolytic varieties.

Adopting first of all the differentiation of streptococci into hemolytic and non-hemolytic types, it may be stated as a broad general finding that (1) hemolytic streptococci are more common in the early stages of infective processes such as gunshot wounds and acute diseases of the jaw; (2) that in chronic diseases of the jaw and in the later stages of wound infection non-hemolytic streptococci are much more common than hemolytic streptococci. Holman's carbohydrate scheme for streptococcal differentiation has been the subject of criticism, by no means unanimously adverse, and as far as carbohydrate fermentation may be used for streptococcal recognition is simple and practical. Henrici modifies the procedure somewhat, omitting lactose. In my own investigations I have followed Holman's classification, using serum broth cultures with Andradad's indicator. Hemolysis was determined by plate method and confirmed by human red cells suspended in broth (Table I). I have frequently found the method of practical importance, and I bear out Holman's original statement that if his method of classification is used it is often possible to determine the origin or site of infection by the type of the group of streptococcal reaction, thus I have isolated a streptococcus from the urine in cases of jaw

TABLE I DIFFERENTIATION OF STREPTOCOCCI HOLMAN



infection, and on comparison of the sugar reactions have identified the streptococci as similar in their reactions to those obtained from the jaw. In other cases, the streptococcus from the urine has not shown the characteristics of those present in the jaw, the reactions suggesting a probable fecal origin, and search for such a cause has demonstrated the presence of a similar type of streptococcus in an infection of the great bowel. It is, I think, a significant fact that in chronic jaw infections and in chronic bone infections following wounds, streptococci of the non-hemolytic variety should predominate. I have shown the various strains of streptococci grouped according to Holman's scheme, isolated from seventy-two consecutive cases of jaw infections, all of which had some clinical symptoms and in which blood changes were present. A little over 50 per cent gave hemolytic streptococci — forty-two cases — while thirty had non-hemolytic streptococci. The morphology the these strains was

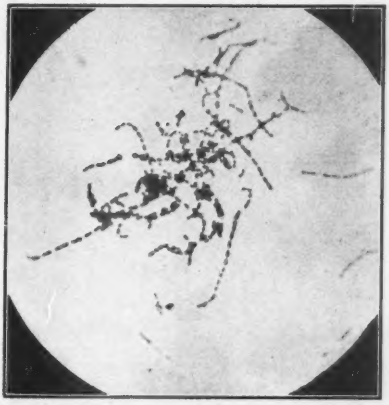


Fig. 46.—Streptococcus jaw, diplo-streptococcal form. Gram. x 1000.

inconstant, but mouth streptococci, when freshly isolated, show a great tendency to bacillary formation. Figures 46 and

47 are two morphological types commonly met with; the organisms showing

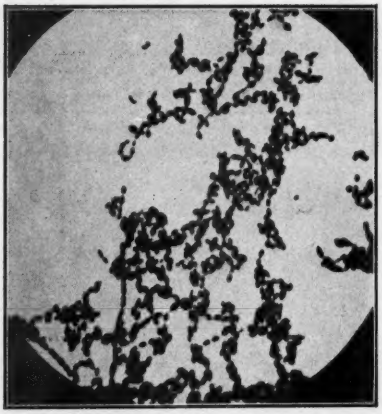


Fig. 47.—Streptococcus jaw, oval and oat shaped form. Gram. x 1000.

the characteristic forms of Figure 47 are usually non-hemolytic.

The histological changes observed as

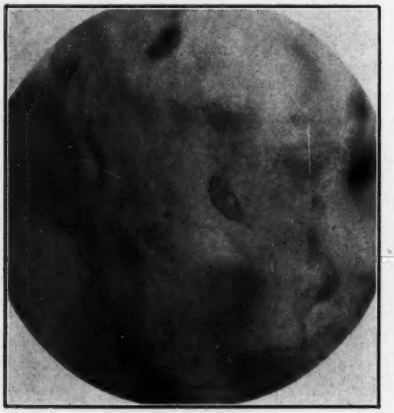


Fig. 48.—Tissue removed at operation, old gunshot fracture, tibia ununited 372 days after injury. Haematoxylin-eosin. x 1000.

the sequelae of persistent jaw infections are similar to the tissue changes following severe infection of bone in gunshot wounds. Thus Figure 48 is a section

Fig. 49.—Tissue removed from root of extracted pyorrhea tooth. Haematoxylin-eosin. x 1000.

of tissue removed from the ununited fragments of a septic fractured tibia 272 days after injury, and Figure 49,

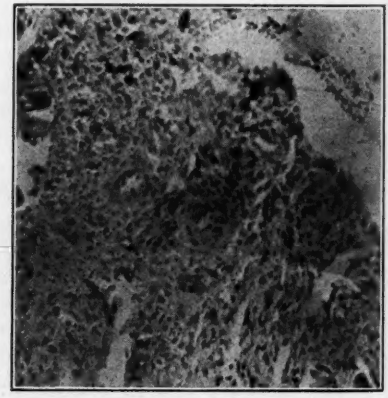


Fig. 50.—Tissue from hypertrophic gum in pyorrhea alveolaris. Haematoxylin-eosin. x 150.

the tissue removed from the end of a tooth in chronic pyorrhea alveolaris.

Figures 50 and 52 are hypertrophic tissue replacing the alveolar process in a long standing case of pyorrhea alveolaris, and Figure 51 the tissue removed from the ununited ends of a septic fractured fibula 120 days after injury. In all four instances non-hemolytic streptococci were isolated from the excised tissue.

During my researches into the infection of war wounds, both recent and

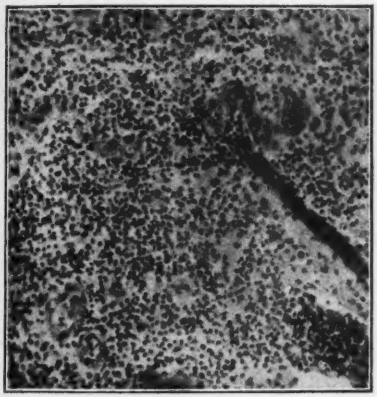


Fig. 51.—Tissue from old septic gunshot fracture of fibula tibia ununited removed at operation. Haematoxylin-eosin. x 150.

Fig. 52.—Tissue removed from alveolar process pyorrhea alveolaris. Stained haematoxylin-eosin. x 100.

latent, I was led to a study of the changes occurring in the blood, and in comparing the blood findings in alveolar infections and in bone infection in septic wounds, certain interesting points of resemblance are evident. It is well known that in certain acute septic infections leucocytosis occurs, and the leucocytosis is commonly regarded as of some diagnostic value. It is usually conceded that the presence of a leucocytosis is in some way associated with the development of immunity as, for instance, the occurrence of leucocytosis in the crisis of pneumonia. Some observers have endeavored to determine if the production of leucocytosis by the administration of chemical substances would increase the immunity of an animal to infectious disease in general. Bullock (3) published some experiments showing that sodium cinnamate, when injected into animals, produced an increase of complement. In later experiments Ledingham and Bullock (16) showed that the opsonic (complement) power of the blood of rabbits was uninfluenced by the injection of autolysed cultures of *many* bacteria or by the injection of nuclein, but that these substances, while producing a decided increase in the number of white blood cells, did not produce as great an effect as many simple chemical substances of which sodium cinnamate is the type.

It is not certain that the establishment of a leucocytosis is an indication of increased immunity of a given animal toward a given disease, but it is an indication of the action of bacterial

TABLE II—BLOOD IN SEPTIC WOUNDS

No.	Red Cells	White Cells	Ratio	Polymorphonuclear A.	F.	G.	To.al	Mononuclear B.	C.	A	F.	Total	B.	Total B. & C.	E.
Normal	5,000,000	6,000	1:800	63%	4%	0%	67%	23%	6%	3740	240	4,000	1,380	1,740	3%
	4,432,000	20,384	1:210	44.5	14.5	1.5	60.5	31.5	5.0	10,600	3,300	14,400	7,500	8,700	1.0
9,946	5,344,000	20,464	1:260	34.0	6.0	0	40.0	57.0	5.5	7,000	1,230	8,250	10,200	10,300	2.5
8,777	5,840,000	29,120	1:200	40.5	36.0	3.0	79.0	10.5	8.5	11,600	10,400	22,900	2,910	5,510	0
8,715	4,832,000	19,968	1:250	60.0	12.5	2.0	74.5	9.0	9.0	11,900	2,380	14,830	1,380	2,420	4.0
8,714	5,128,000	19,760	1:260	49.5	20.5	1.0	71.0	14.5	8.5	9,750	4,070	14,000	2,832	4,580	1.0
10,364	4,208,000	18,304	1:230	53.0	21.5	2.0	76.5	11.5	10.0	9,700	3,820	14,000	2,110	3,940	1.0
10,363	3,646,000	12,688	1:300	37.0	24.0	1.0	62.0	28.0	9.5	4,700	3,200	7,850	3,520	4,900	0.5
	4,960,000	16,828	1:300	62.5	11.5	0.5	73.5	16.5	8.5	11,050	1,860	12,320	2,700	1,430	0.0
11,251	4,928,000	14,164	1:340	51.5	6.0	0.0	56.5	34.5	7.5	7,300	850	8,000	4,880	5,950	0.5
7,308	6,016,000	25,792	1:220	54.0	6.5	1.0	60.5	31.5	2.0	13,950	1,680	15,530	8,150	8,900	1.5

TABLE III—BLOOD IN PYORRHEA ALVEOLARIS. ARTICULAR RHEUMATISM

No.	Red Cells	White Cells	Ratio	Polymorphonuclear A.	F.	G.	Total	Mononuclear B.	C.	A	F.	Total	B.	Total B. & C.	E.
Normal	5,000,000	6,000	1:800	63%	4%	0%	67%	23%	6%	3740	240	4,000	1,380	1,740	3%
12,705	6,072,000	17,180	1:340	47.0	6.0	0.0	53.0	32.0	14.0	8,080	1,000	9,000	5,460	7,500	0.5
10,094	3,232,000	12,688	1:270	61.0	4.0	0.5	65.5	26.5	6.5	7,740	500	8,260	3,330	4,180	1.0
12,787	6,120,000	12,200	1:500	49.5	10.0	1.0	60.5	24.5	12.5	6,900	1,220	7,320	2,900	4,490	1.5
12,809	6,702,000	12,190	1:500	48.5	7.0	1.5	57.0	23.5	16.0	5,920	855	6,960	2,840	3,510	2.0
10,886	6,176,000	11,648	1:550	45.0	18.0	0.5	63.5	25.0	8.0	5,250	290	7,350	2,950	3,850	1.5
11,931	6,576,000	11,960	1:540	49.0	17.0	2.0	68.0	24.0	4.5	5,860	202	8,140	2,880	3,550	2.0
11,899	6,224,000	10,580	1:600	61.0	9.0	0.0	69.0	24.0	5.0	6,450	950	7,300	2,520	3,500	0.5
13,389	5,440,000	13,020	1:400	54.5	7.5	0.0	62.0	24.0	11.0	7,100	980	8,100	3,130	4,560	2.0
11,867	6,896,000	17,020	1:400	46.0	4.0	0.0	50.0	36.5	12.5	7,850	682	8,520	6,250	8,350	0.5
11,516	6,424,000	13,936	1:500	55.5	9.0	0.0	64.5	26.0	0.5	7,730	1,250	8,990	3,620	4,550	1.0

TABLE IV—BLOOD IN PYORRHEA ALVEOLARIS. PERI-ARTICULAR RHEUMATISM

TABLE IV—BLOOD IN PYORRHEA ALVEOLARIS. PERI-ARTICULAR RHEUMATISM

No.	Red Cells	White Cells	Ratio	Polymorphonuclear A.	F.	G.	Total	Mononuclear B.	C.	A	F.	Total	B.	Total B. & C.	E.
Normal	5,000,000	6,000	1:800	63%	4%	0%	67%	23%	6%	3740	240	4,000	1,380	1,740	3%
12,321	6,704,000	8,510	1:800	49.5	13.5	0	63.0	23.5	9.5	4,200	1,150	5,350	2,000	2,800	4.0
12,991	5,620,000	8,050	1:700	33.5	16.5	0	50.0	38.5	8.0	2,640	1,280	4,020	3,400	3,680	2.0
12,075	5,552,000	7,130	1:800	26.5	26.9	1.5	54.0	34.5	8.5	1,880	1,850	3,830	2,420	3,120	2.5
9,410	3,752,000	5,824	1:800	63.0	1.5	0	64.5	33.5	1.0	3,700	620	3,820	2,000	2,100	0
13,143	5,210,000	4,930	1:1000	28.5	12.0	0	66.0	24.5	8.0	1,499	590	3,230	1,180	1,570	1.5
12,555	7,312,000	6,300	1:1200	34.5	12.0	1.0	47.5	22.0	14.5	2,140	755	2,930	1,390	2,230	13.5
12,611	5,200,000	5,750	1:1040	41.5	8.0	0	49.5	32.5	10.0	2,390	460	2,840	1,853	2,420	7.0
12,943	6,460,000	4,360	1:1600	53.0	12.5	0	65.5	23.5	9.5	2,300	545	2,840	990	1,440	1.0
12,882	4,932,000	3,620	1:1600	26.5	49.5	7.0	74.0	10.0	11.5	950	1,450	2,660	362	774	2.0
12,886	3,980,000	3,680	1:1400	38.5	24.5	3.0	66.0	19.5	12.5	1,410	900	2,430	710	1,150	0
10,960	6,280,000	3,536	1:2000	44.5	25.0	0	69.5	21.5	5.0	1,560	870	2,400	730	910	2.0
8,349	2,896,000	3,120	1:900	39.0	12.0	0	51.0	41.0	4.0	1,220	376	1,600	1,280	1,420	2.5

TABLE V—BLOOD IN PYORRHEA ALVEOLARIS

No.	Red Cells	White Cells	Ratio	Polymorphonuclear A.	F.	G.	Total	Mononuclear B.	C.	A	F.	Total	B.	Total B. & C.	E.
Normal	5,000,000	6,000	1:800	63%	4%	0%	67%	23%	6%	3740	240	4,000	1,380	1,740	3%
12,871	5,259,000	6,900	1:750	43.0	10.5	0	53.5	29.5	10.5	2,970	690	8,640	2,000	2,000	3.5
12,936	6,780,000	5,980	1:1100	39.5	11.5	0	51.0	28.5	17.5	2,340	660	3,400	1,700	2,740	3.0
13,152	6,120,000	5,960	1:1000	46.0	6.5	0	52.5	38.5	6.5	2,920	387	3,100	2,340	2,680	2.0
12,502	5,856,000	4,830	1:1100	35.5	29.0	0	64.5	26.5	7.0	1,710	1,400	3,100	1,280	1,660	1.0
12,577	5,440,000	5,290	1:1000	47.0	6.0	0	53.0	31.0	10.0	2,480	310	2,800	1,640	2,135	3.0
12,862	5,762,000	4,120	1:1200	54.5	11.5	0	66.0	21.5	6.5	2,260	450	2,710	840	1,150	5.0
9,020	4,352,000	3,952	1:1000	51.5	10.0	0	61.5	27.5	8.5	2,000	400	2,460	1,000	1,480	0
10,512	6,304,000	3,952	1:1500	47.5	1.5	0	49.0	44.5	5.5	1,850	415	1,940	1,760	1,980	0.5
13,036	7,630,000	4,150	1:1900	33.0	11.0	0	44.0	38.0	14.0	1,350	450	1,815	1,552	2,140	3.0
10,142	4,864,000	2,496	1:1600	38.0	15.0	0.5	53.5	33.9	12.0	950	370	1,330	820	1,120	0.5

proteins on the blood. Further, a diminution in the number of white blood cells (leucopenia) is known to occur after the injection of toxic substances, and among such toxins are the toxins of diphtheria, tetanus, and snake venom.

In examining the blood of infected wounds I have found that both categories, namely leucocytosis and leucopenia, are observed, and that the presence of leucopenia or leucocytosis bears a relation to the clinical character of the disease. The enumerations were made by a slight modification of the Arneth count, namely, a differential count of the polymorphonuclear leucocytes in addition to the ordinary enumeration of the mononuclears, eosinophils, and other white cells (Table II).

A similar series of enumerations has been made in a very large number of cases of latent and obvious infection of the alveolar process of the jaw and I have further classified these counts with relation to the general symptoms from which the patient was suffering and have placed them in three main categories: (1) pyorrhea alveolaris with articular rheumatism associated with bone involvement—"atrophic arthritis" and "hypertrophic arthritis" (Table III); (2) periarticular rheumatism without gross bone changes and with various pathological processes involving the periarticular tissues, often classed as "fibrositis" (Table IV); (3) pyorrhea alveolaris with various toxic symptoms, some of an indefinite kind which are often classed together as "secondary neurasthenia," cardiac, vascular, and intestinal (Table V).

The enumerations are made on slides stained by Giemsa, two hundred cells being counted in the differential counts. The actual number of white cells in each category is calculated from the percentage and the total count is calculated in round numbers. A very striking similarity is shown between the septic wounds of severe infection (Table II) and cases of articular rheumatism with bone involvement. Both cases show total leucocytosis, mainly falling on the polymorphonuclear cells, a general increase in the immature cells or "rod nuclears" on the Arneth's count, and a tendency for a general increase of the mononuclear cells. In both cases there is a low ratio of white to red cells. In the other two classes (Tables IV and V) there is a general tendency to a decrease in the polymorphonuclear cells of the mature type with an increase in the immature cells; moreover, in fibrositis of severe type, the mononuclear cells also show a tendency to increase (Table IV). In those cases with neurasthenic symptoms the immature cells, altho variable, show a general tendency to increase. Low readings are extremely uncommon, but the total number of polymorphonuclear cells is, on the whole, below normal. The mononuclear cells show more variability in their counts and, on the whole, appear about normal. The tables given are characteristic of the various types and are taken at random from a large number of cases, and I think show that the more severe mouth infections have more than a superficial similarity to bone infection in other regions of the body, and that the less severe cases of latent oral disease show certain blood changes in common with the more severe type, but that this change, altho similar, is less in degree and is modified along defined pathological channels.

The limits of my subject have precluded any discussion of the etiology of jaw infections in general or of the commonest cause of latent infection, or subinfection, namely, pyorrhea alveolaris. However, while preparing the present paper, I had the opportunity of

seeing the recent experimental work of Professor and Mrs. Mellanby, on rickets, and I was so impressed with the results they had obtained that I requested and obtained their ready consent to photograph certain sections of the jaws of puppies which had been fed on certain diets free from "fat soluble A." I have frequently seen hypertrophic disease of the gums and early pyorrhea

EXPERIMENTAL RICKETS
BONE CHANGES

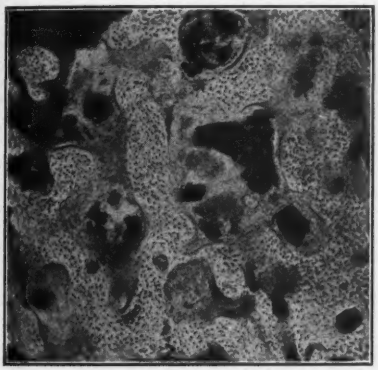


Fig. 53.—Normal puppy 177. (Diet A + cod liver oil). Section of bone body of jaw. Borate-carmine. x 50.

alveolaris in children, especially girls at boarding schools, the cause of the disease being very difficult to understand. Many cases of pyorrheal trouble commence in quite young people; in a large proportion of adults under thirty who are the subjects of pyorrhea a history of bleeding gums while at school may be elicited.

Professor and Mrs. Mellanby (17) fed their two series of puppies on a diet of protein and carbohydrate; diet "A" the necessary fat factor differed in the two cases; to the one cod liver oil was given as fat, to the others linseed oil. The results on the development of the teeth, jaws, gum tissue, and peridental membrane is seen in the illustrations. The sections were prepared by Weil's method and stained with carmine.

Fig. 54.—Puppy 177. (Diet A + linseed oil + extra milk). Section of bone body of jaw, which was much thicker than the normal animal. Irregular, poorly, calcified bone. Borate-carmine. x 50.

Fig. 55.—Puppy 176. (Diet A + linseed oil only). Section of bone body of jaw much thickened and alveolar margin slightly irregular and roughened. Irregular osteoid tissue bone structure entirely altered. Borate-carmine. x 50. All these specimens were prepared by Weil's process and I am greatly indebted to Professor and Mrs. Mellanby for permission to photograph and use these illustrations.

Figure 53 is the bone of the jaw of a puppy fed on diet A plus cod liver oil. Bone cells and marrow are normal. Figure 54 is from the jaw of a puppy fed on diet A plus linseed oil but with some extra milk. Figure 55 is from the jaw of a puppy fed on diet A plus linseed oil only. Note the irregular osteoid tissue in the absence of any typical bone structure. The whole structure is of a different character from that of normal bone. Figure 56 is a section thru the carnassial tooth of the normal animal (A plus cod liver oil). Figure 57 is a section thru the carnassial tooth of a puppy fed on diet A plus linseed oil and extra milk. Figure 58 is a section thru the carnassial tooth of puppy fed on diet A plus linseed oil.

Figure 59 is portion of the denture to show irregular dentin and peridental membrane.

On comparing the illustrations, the vitamine deficiency has produced: (a) irregular and feebly calcified dentin; (b) irregular and ill-formed enamel which stains with the carmine; (c) osteoid tissue forming the alveolar process, much thickened; (d) badly formed gum tissue, very deficient toward the tooth; (e) a very weak alveolar-gum junction.

Apart from the extreme importance of this observation on the etiology of rickets, the possible explanation of lowered resistance to bacterial inroads at the dentoalveolar periosteum angle is

EXPERIMENTAL RICKETS
TOOTH CHANGES

Photograph of Professor and Mrs. Mellanby's specimens.

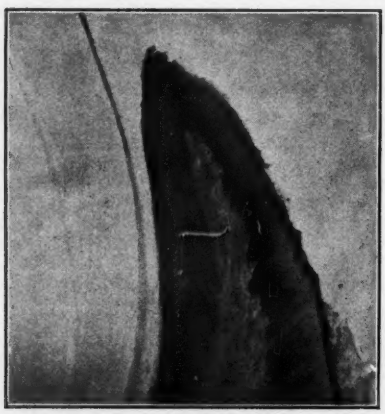


Fig. 56.—Section thru carnassial tooth of puppy (179) fed on normal diet. A, which contains no "fat soluble A" vitamine; cod liver oil given to supply this vitamine. Normal growth of enamel denture, and bone of alveolar process. Note especially well developed gum tissue covering bone, both on outer side and next to tooth. Borate-carmine. x 50.

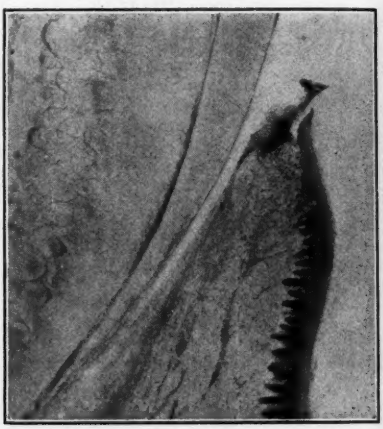


Fig. 57.—Section thru carnassial tooth of puppy (177) fed on normal diet A plus linseed oil which altho supplying fat contains no vitamine ("fat soluble A"). Irregular development of dentin which is full of irregular interglobular spaces; thin, badly formed enamel; badly developed and hypertrophic alveolar process, and thin ill formed gum tissue which is almost absent on the side next the tooth. Borate-carmine. x 50.

at once suggested, and moreover some color is given to my suggestion that pyorrhea infection may be a late manifestation of early deficiency in vitamine.

To summarize the various methods of diagnosis which have been outlined we may conclude:

1. That a careful comparative study of bone disease in other parts of the body throws much light on the septic infections arising in and about the jaws.

2. That radiographic examination is essential to the diagnosis of any case of jaw infection, both to determine the extent as well as the character of the disease.

3. That blood examinations show that the severer infections have a common blood picture with bone disease of streptococcal origin generally and that the less severe infections have a blood picture linking them with the graver conditions.

4. That removal of the "septic focus" does not connote tooth extraction in all cases and must be regarded as an expression of failure in early diagnosis and treatment.

5. That early food deficiencies may have an etiological relation to pyorrhea alveolaris.

Fig. 58.—Section thru carnassial tooth of puppy (176) fed on normal diet A plus linseed oil. Irregular development of enamel which has taken up the stain; ill formed dentin, badly developed irregular gum tissue and alveolar dentoperosteal angle. Borate-carmine. x 50.

Fig. 59.—Section of dentin cementum and pulp from last Figure (177) showing interglobular space in dentin and badly formed dento-alveolar-periosteum. Borate-carmine. x 50.

The equilibrium of the tissues and the infecting organisms is of prime importance, as if the blood is sharing in maintaining the balance, certain changes are found to take place, small in amount, but pointing to some exciting cause, and too great caution cannot be taken in the interpretation of individual blood counts without the due consideration of collateral evidence.

When oral infection is in fact producing remote effects, evidence is forthcoming when proper radiographic

hematological, bacteriological, as well as clinical data are duly examined.

And that finally, without a careful discrimination based on such knowledge, it is not justifiable to postulate oral infection as the cause of a complaint, or still worse, proceed to radical surgical interference on insufficient date.

TABLE II

Osteomyelitis in war wounds.

(i) Total increase of all white cells, with low ratio.
(ii) Especial increase of immature polymorphs, often ten times normal, even when total polymorph count only 3-5 times normal.
(iii) Mononuclear cells increased variably, most in worse cases.

TABLE III

Pyorrhea in polyarticular arthritis.

(i) Total increase of all white cells, with low ratio.
(ii) Especial increase of immature cells, often very high. Increase of A. less pronounced than F.
(iii) Increase of mononuclears, more in most severe cases.

TABLE IV

Periarticular arthritis and general fibrositis.

(i) Total decrease white cells with high ratio.
(ii) Increase of juvenile cells, often extreme.
(iii) Decrease of multi-nuclear cells.
(iv) Increase of lymphocytes in most severe cases.

TABLE V

Pyorrhea with variable symptoms.

(i) Total decrease white cells with high ratio.
(ii) Slight increase in juveniles, less than other classes. (General decrease).
(iii) Decrease in polynuclears in all cases.
(iv) Always high mononuclears in severe cases.

EXPLANATION OF LETTERS IN TABLES

A, Polymorphonuclear leucocytes, polynuclear.
F, Polymorphonuclear leucocytes, horse shoe nuclear.
G, Polymorphonuclear leucocytes, rod nuclear.
B, Lymphocytes.
C, Large mononuclear.
M, Mast cells.
E, Eosinophiles.
D, Basiophiles and aberrant cells.

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Reminiscences of Forty-five Years in Dentistry

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VI

AFTER graduation I opened an office in Collingwood on the Georgian Bay. Here I practiced from the spring of 1881 to the fall of 1884, getting my first real start in life and forming friendships which have lasted on down thru the fleeting years. I shall always think of the people of Collingwood with sincere affection, because of their cordial reception, and the encouragement they gave me from the start. I soon had a full practice, and began to experience the glow of satisfaction which comes to a young man when he first tastes the sweets of success. Of course my start in Collingwood was hedged about by the limitations of financial stress. I had borrowed money to go to college, and had to borrow more to procure a few meager necessities with which to open an office. I am tempted at this point to say something about my equipment so that the young men of the present may compare it with the sumptuous offices to which many of them aspire. I am a believer in a well-equipped office, if a man can afford it, but I do not believe in a young man loading himself down with a heavy debt just to make an ostentatious show, as too many of them are doing today. A practice can be built without an expensive office provided everything about it is kept clean, neat, and attractive. It is the personality and service of the dentist that counts more than a mere showy office. I do not submit my humble outfit of forty years ago as a sample for the young man of today, but I cannot forego the impulse to warn our recent graduates against contracting heavy obligations for the purpose of display. Too many of them come to grief and register an ignominious failure where they might have succeeded if they had started in a less pretentious way, and avoided the incubus of too burdensome a debt.

I had such an aversion against owing money that I began practice even without a dental engine, doing all my operating with hand instruments for the first few months. I had no dental chair, and my first work was done in an ordinary rocking chair with a block of wood under the rocker to keep it tipped back. When my landlord saw what I was doing he insisted on my accepting a loan of forty-five dollars with which to buy a barber chair, and with this luxurious outfit—it *was* luxurious in my eyes—I began to hew my way out of debt. I never worked with greater satisfaction in my life than I did during those few years in Collingwood, not even when in later days it was my privilege to serve many interesting people in a large city practice.

I did not rest till I was free from debt, and when that was accomplished I breathed more easily, and yet I was fortunate in not making the fatal mistake that so many have made. When success came I did not lessen my efforts but

nursed my practice as carefully as ever, realizing even at that early day that the most critical time in the career of any man is when he seems most secure in his position. I have always been impressed with the fact that to maintain success is almost as difficult as it is to achieve it, and this is true not only of men, but of institutions, and of nations. Rome fell when she seemed supreme—an outstanding instance among myriads of others, before and since, of the danger of assuming superiority, and resting on a reputation that has already been made.

I had of course many amusing experiences, as does every man who conducts a practice, particularly one where patients come occasionally from the back settlements. One day a lanky, weazen sort of man entered the office, and began walking around the room, looking up at the pictures on the wall, and examining the various articles of furniture with the greatest interest. I had spoken to him when he came in, but he merely answered with an indifferent nod, and paid no further attention to me in his apparent absorption with the contents of the office. I finally asked him if there was anything I could do for him, and he answered, "No"—still staring around the room. I began to think I had encountered a benevolent species of lunatic, and watched him to await developments. After surveying everything to his heart's content he heaved a sigh of wonderment, and then in the most offhand manner remarked: "I've the funniest tooth you ever seen." I asked him what was the matter with it. "Nothin'," he replied. "Well—does it ache?" I ventured. "No," said he. "Why is it so funny?" I asked. "Well sir, sometimes it seems as if it would take the top of my head right off. It never hurts in any way, but it nearly takes the top of my head off." I suggested that we had better take a look at it, and led the way into the operating room. The whole procedure was apparently so new to him that it was plain he had never been in a dental office before. I found a molar decayed beyond repair, and told him that it should be taken out—warning him that it might hurt considerably to have it extracted. It was difficult to make any impression on his mentality, and I concluded that the proper thing was to take the tooth out and be done with it. When I took hold of it with the forceps he was the most surprised man in North America, and let out a whoop that would have turned a Comanche Indian green with envy. The instant I got the tooth out he seized his jaw with his hand and rocked back and forth moaning in the most pitiful way. I was distressed beyond measure, and tried to console him, when all at once he shrieked louder than ever, and exclaimed in terror, "It's bleedin'—it's bleedin'!" After I had succeeded in quieting him, he asked to see the tooth. Taking it in his fingers and examining it curiously a moment he scratched the darkened enamel with his finger nail and said: "The little thing is all furred up, isn't it? It's black as a toad's back." Which, after all, was a very accurate description of the tooth, as he held it in his hand.

Another incident occurred one day which started out rather disagreeably, but which, so far as I was concerned, ended with much amusement. An Irishman for whom I had inserted an upper denture came in the office, and began to complain about the teeth. I tried to ascertain what the difficulty was, but with little success. All he would say was: "They jist don't feel right," and began to take me to task for not doing better work. I had a young dentist friend visiting me, and he listened to the conversation. When unable to control himself he turned to the Irishman and said: "You are the most unreasonable man I ever heard talk. What's wrong with the teeth anyhow?" Instantly the air was electric. The Irishman diverted his attack from me to my friend, and the merry war was on. I have seldom heard two people call each other a more choice assortment of

names than they did, and I was soon entirely out of consideration so far as the resultant situation was concerned. After they had exhausted a most prolific vocabulary on each other, the Irishman turned to me with a mollified look in his eye and said: "Anyhow, Dr. Johnson, you're a *gintleman,*" and walked out of the office. I never heard further from him or his teeth, which leads me to remark that "a friend in need is a friend indeed."

Two very pleasant incidents have come to me in recent months as an aftermath of my practice in Collingwood. In February, 1921, I was motoring with my friend Dr. B. B. McCollum from Los Angeles to San Diego. On the way down I mentioned the fact that I had an old friend in San Diego—an artist—whom I wished to see. We drove to his studio, and the first question he asked me was: "Did you get my letter?" I said I had not. "Well," said he, "when you get home the letter will be there, and in it you will find a piece of gold, the history of which is this: The other day I was having one of my front teeth cut off for a porcelain crown, and as it came off I told the dentist to break the tooth open and let me have the gold filling that was in it. I said I wanted to send that filling to Dr. Johnson, because he inserted it forty years ago in Collingwood, Ontario."

I have that filling in my possession today, and needless to say it will remain there.

In September, 1921, I received the following letter from A. E. Brown, County Physician and Superintendent of the Lake County General Hospital of Waukegan, Illinois:

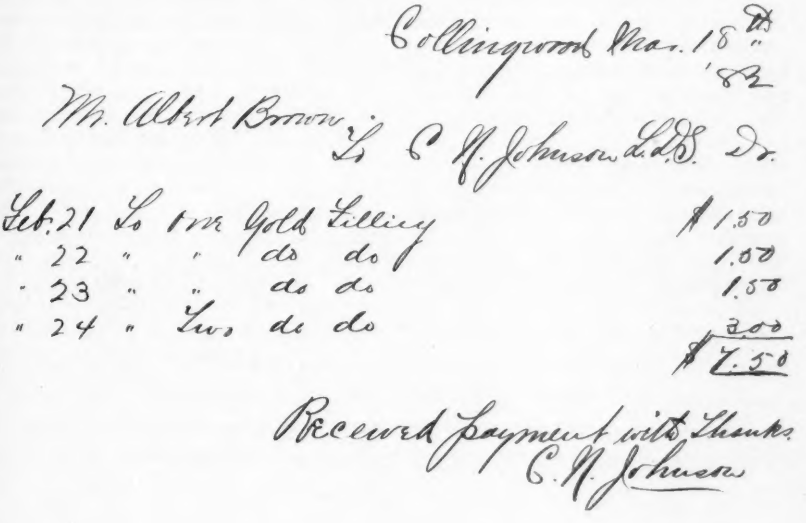
"My Dear Doctor:

In housecleaning I unearthed the enclosed. I thought you might be interested in knowing that there are those living who knew you when you worked with a mallet and chisel. These fillings were taken from my mouth about one year ago.

Sincerely,

A. E. Brown."

The inclosure was a bill I had rendered him in March, 1882, and it is herewith reproduced.



Collingwood Mar. 18th 82

Mr. Albert Brown
To C. H. Johnson L.D.S. Dr.

Feb. 21	To 1 Gold Filling	$1.50
" 22	" " do do	1.00
" 23	" " do do	1.50
" 24	" Two do do	3.00
		$7.50

Received payment with Thanks
C. H. Johnson

Dr. Brown had written on the back of the bill: "Dentists were not highwaymen in the good old days." And I think my readers will agree with him.

When I showed this bill to a dentist friend of mine, he remarked: "You would probably be ashamed to have anyone know what your fees were in those days." On the contrary I take greater pride in showing that bill than I would to show a bill for the largest fee I ever received.

It must be remembered that in those days all kinds of commodities were lower than they are today. Professional fees in that section were never high, and there was another factor which entered into consideration. A large percentage of the fillings inserted had been of amalgam, and it became my ambition to introduce gold more extensively. To do this I was obliged to make the fee as reasonable as possible, until I had built up among the people an appreciation of the value of gold fillings. It was in a certain sense pioneer work, and I have no regrets over the ultimate outcome. Before I left Collingwood I was inserting gold in a relatively large percentage of cavities, and doing it at a remunerative fee.

I want to see my profession well compensated for their services, because I know of no work more exacting than this; and yet when I hear of some of the fees that are being demanded today, I blush lest the charge of profiteering may reasonably be brought against us. And I am going to be frank enough to ask the blunt question—which is the more commendable, to insert gold fillings for $7.50 which last for thirty-eight years, or to charge $750.00 for a piece of work which is so ill-advised in construction that it goes to pieces in a year or two? I have seen the latter happen more than once. The prevalent commercialism of the age has not altogether escaped the professions in its mad career, and it would sometimes seem as if that commendable entity known as professional conscience had been administered a lethal dose.

Speaking of pioneering reminds me that I made my first venture in the literary field while in Collingwood. I found such a dearth of knowledge concerning the teeth that I issued a pamphlet for free distribution, the title of which—if I recall correctly—was *The Teeth, and How to Preserve Them.* I have not seen a copy of that pamphlet for many years, but crude and amateurish as it was, it did a more definite good in the community than I should care to claim for any similar piece of writing I have done since. The problem of educating the people as to the significance and care of the teeth is still with us, and yet we have made great progress since those days. There is a wider range of knowledge on these subjects and a more general appreciation of the value of dental service. And each man in his own community can do much to extend this knowledge, and increase this appreciation if his daily attitude toward his work and his patients is of the right kind. This obligation of carrying the proper message to the people and serving them in such a way that they are not only benefited themselves but that they will extend the message to others is second only to the obligation which a dentist owes to his family.

(*To be continued*)

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Editorial Department

THE NEW RECRUITS

At this season of the year when so many of our young men are finishing their college course and entering on the practice of their profession, it may be well to point out some of the things which the profession has in store for them. It is quite impossible for the average young man to realize the full significance of the professional and moral obligation which he assumes when he bows his head and accepts a diploma on commencement day. The uppermost thought in his mind is

one of triumph—triumph at having successfully met the requirements of the college and received the indorsement of the faculty. He fancies that he has attained the summit—that it will be all downhill from this on—that all he has to do is to hang up his coveted diploma and reach out his hand for success. But the dawning of a sterner day will come, when he will be confronted with the stubborn realities of life—a life that he was never able to visualize in the palmiest of his college days. He will be brought face to face with the sober fact that the real test of human mettle is the alembic of practical experience. How could he know that in everyday practice the conditions are entirely different from the days when he had a demonstrator constantly at his elbow? No matter how many times his college professors have warned him of this he cannot see it till he is actually in contact with it. The experience and example of other men are as nothing to him in this period of his professional adolescence. He must learn for himself—and learn he does.

Sometimes the lesson is so severe that it leaves him limp and disheartened, and yet it is only by this apparently bitter experience that he can finally grow into the full stature of professional manhood. And let it be said that today, with the rapid development of the profession, the achievement of professional manhood is something well worth while. It is a goal toward which the young man should strive, no matter at what cost of personal endeavor or sacrifice. Dentistry today holds more in the way of reward than at any time in its history. The new recruit comes into a profession which at this time commands respect. It is rapidly climbing to a parity with any other of the learned professions, and thus the recent graduate has a sacred obligation forced upon him at the very threshold of his professional career. He has the obligation of doing his share in a manly and whole-hearted way to sustain the better traditions of his profession, and to add, even in the smallest possible way, to the betterment of his calling.

Few young men come into the profession with a sufficiently exalted conception of their duty to their profession or to society at large. Their chief concern naturally relates to earning a livelihood, and it is not till later in life when the mellowing influence of an association with human suffering pours its beneficent oil over their consciousness that they come fully into a realization of the highest function of following a professional career. This is a matter of growth—not of sudden inspiration. It must be knit stitch by stitch into the fiber of their professional being.

Fortunate the young man who early in his experience begins to absorb the essence of what true professionalism means! It is out of this realization that comes the greatest happiness in professional life,

and the greatest achievement toward that unselfish service to humanity which brings the surest and sweetest reward.

Dentistry offers opportunities today that it never did before. If the hill is steep and the road hard, there is a commensurate reward at the top, and every young man has it within him to achieve at least a modicum of this reward if he will only apply himself. And he must remember that the road is not so hard today as formerly. The path has been worn smoother by the footsteps of those who have gone before—by the tread of the pioneers, and the pathfinders. He should be thankful for this, and in his gratitude for the privilege he enjoys he should dedicate himself in all seriousness to a life of professional service and sacrifice, which shall leave his chosen calling a niche higher in the realm of human endeavor, and more nearly perfect in its ministration to the ills and sufferings of his fellow man. If he but adds one jot or tittle to the sum total of human comfort or human happiness, his life may be called a success, and he may lay down his burden at the end of the winding road with a sense of having contributed his share to the betterment of the world in which he lived. This is a prospect which no young man can afford to ignore, and it is commended to the attention of every one of the recent graduates who are entering the profession at this time.

INTERNATIONAL DENTAL FEDERATION (F. D. I.)

The aims and objects of the *Federation Dentaire Internationale* are not as well understood by the rank and file of the profession as they should be. It was organized with the idea of bringing cohesion among the professional interests of the different nations, and was the outcome of the Third International Dental Congress held in Paris, France, in 1900. Heretofore there had been no authorized international body existing in dentistry which could function between the various international congresses, and one of the provisions in its statutes says: "The International Dental Federation is a permanent international body existing in the interim between International Dental Congress."

The amount of good which this organization accomplished between 1900 and the breaking out of the world-war can never be estimated. It was the one tribunal to which organized dentistry the world over could appeal, the one link to bind inseparably the professional interests of the various nations.

Unhappily the breaking out of the war in 1914—just at the time a meeting was being held in London—interrupted the beneficent function of this organization, and while interest in the body never relaxed on

the part of those most vitally concerned in its welfare, it was not till September, 1921, at The Hague, Holland, that a meeting was finally held. This meeting was largely for conference, with the idea of definitely determining the future activities of the Federation, and to decide when and where the next regular meeting should be held. The following nations were represented: France, Belgium, Italy, Holland, Spain, Denmark, Sweden, Norway and the United States. It was decided that the International Dental Federation should continue its activities as an international body, and that the formal meeting should be held in Madrid, Spain. The date of this meeting has been set for August 28, 1922.

It will thus be seen that the great value of this organization to the dental professional interests of the world is not to be lost, and the probability is that it will be even more active and influential in the future than it has been in the past. It would be difficult to conceive of the greatest possible professional advancement the world over without this organization. It is the leavening and harmonizing influence between the nations—the one integral and constant entity which binds the profession together at all times.

The President of the F. D. I. is Truman W. Brophy, of Chicago, and the secretary, Florestan Aguilar, of Madrid, Spain. The dentists of America owe their allegiance and support to this international body, not only because the Federation has honored one of their own number by assigning to him the highest office in its gift, but because of the objects and aims of the organization, which has for its purpose the betterment of dentistry in every quarter of the civilized world.

IN MEMORIAM: WILLIAM VAN BERGEN AMES

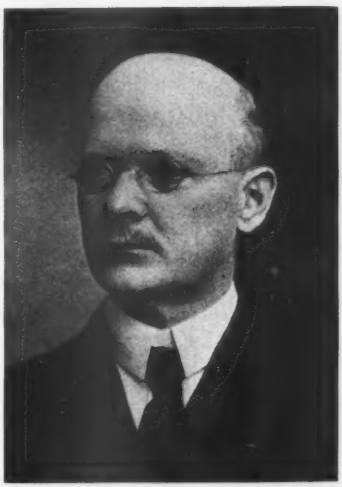
William Van Bergen Ames was born May 27, 1859, at Attica, Ohio. His father was an eminent physician of that state. The family moved to Fremont, Ohio, where the son attended public school until he entered Notre Dame University, South Bend, Indiana. When eighteen he was sent to the mountains of northern California on account of ill health, where he entirely recovered. He entered the Ohio Dental College at twenty years of age. A prize was offered by the College for the best bridge of four teeth; he made the best for six teeth. He took the gold medal, and also won a gold medal for superior work in chemistry, thus giving early promise of his splendid achievement later in the perfection of dental cements. He was graduated in 1880, and located in Chicago in 1881.

For a time he taught in the Chicago College of Dental Surgery, and in 1891 he with four others organized Northwestern University Dental School, where for several years he was the professor of prosthetic dentistry. Dr. Ames was a member of the Chicago Dental Society, the Odontological Society of Chicago, the Illinois State Dental Society, and the National Dental Association. He was one of the original inventors of the gold inlay, and he also made improvements in dentures and

prosthetic appliances generally. In addition to this his liking for chemistry led him to the study of cements, but it was not until hs eyes began to fail for close dental work that he was induced to put his perfected product on the market.

He made a great success of this enterprise, and gradually was able to travel and see much of the world, visiting all parts of the United States, and much of Europe, Panama, Jamaica and Cuba. As soon as he achieved affluence he began to quietly lend a helping hand to others to add to their pleasure and comfort, and was instrumental in educating a number of young people. These benefactions he kept closely to himself.

In 1913 he purchased a farm of eighty acres near Lake Forest, Illinois, to satisfy his longing for country life, and from time to time he added to this until at the date of his death he owned eight hundred acres. It had not been his intention to farm extensively but merely to enjoy his broad acres and tree land; but when the war came it brought the plea for every farmer to cultivate the land to its full limit. This Dr. Ames did, and the problems presented by war-time farming did much to undermine his health. He loaned one hundred and ninety acres to the Women's Land Army of America, and this was cultivated for war purposes by the "farmerettes."



WILLIAM VAN BERGEN AMES, D.D.S.
1859–1922

In the winter of 1919-1920 he went to Arizona to escape the rigors of the Chicago climate, and in 1921 he built a large home near Chandler, Arizona, where he expected to spend his winters and entertain his friends. In January, 1922, while at his Arizona home he became quite ill, and passed away in February.

In 1895 Dr. Ames married Virginia Gilmer, daughter of Dr. T. L. Gilmer, and they, in their common interests, enjoyed a peculiarly happy life. The sympathy of a host of friends goes out to Mrs. Ames.

The contributions of Dr. Ames to dental literature covered a period almost from the time of his graduation down to 1917. He wrote on a variety of subjects, but in later years his chief interest lay in a study of the different filling materials. He gave many clinics particularly during the early days of inlay work. He left the profession vastly his debtor, not only for what he wrote but for what he did, and his lovable personality will linger long in the memory of his many friends.

Modest in his demeanor, retiring almost to the point of diffidence, but loyal and steadfast in his friendship, he has left an example to all those who were fortunate enough to know him well, that will not soon be forgotten. In a sense he was cast in a heroic mold. He bore patiently and without protest the many ills of life; and there were very few, even among his numerous friends, who knew of his illness till the final summons came. He was in love with life, and particularly with the great out-of-doors. It had been his wish to wave his last farewell to earth from his beloved Briar Ridge Farm in Illinois, but lacking this he was grateful for the

privilege of spending his final days at his desert home where he could watch the stars, hear the songs of the mocking bird at his window, commune with the mountains, and see the glorious sunsets of the west.

Across the threshold where myriad feet have trod before, he left this "bank and shoal of time," and bequeathed to his family and friends a memory which will live on into the vast receding years. The world is better today because Dr. W. V. B. Ames lived in it, and it is fitting that, out of the fullness of their hearts, his friends bring to his bier their wreath of immortelles.

THE ANTISCORBUTIC PROPERTY OF FOODS

Sherman and Smith, in their recent competent review of the present status of the vitamines in science and practice, believe it safe to say, on the basis of our present knowledge, that a dietary selected to make the best use of our ordinary staple foods will give little occasion to purchase vitamines in any other form. Moreover, they indicate that it is not necessary to give any greater anxiety to the vitamines than to some other factors which enter into our present conception of nutritive requirements and food values. "When research in this field," they say, "brings us quantitative knowledge as to the efficiency with which vitamine C can be preserved in our staple fruits and vegetables when canned or dried, comparable with our present knowledge of the good preservation of vitamines A and B in cooked, canned or dried milk, it will become possible to plan food supplies, even for conditions in which fresh food cannot be obtained, with a similar degree of definiteness with reference to vitamines as to other nutritional needs."

It has become common knowledge that not only the traditionally used fruit juices but also many greenstuffs have considerable antiscorbutic potency, that is, they contain the so-called vitamine C. Some of the highly potent antiscorbutic foods, such as cabbage, turnips and tomatoes, are usually so readily available and so well tolerated that one need not search among more expensive products for protective material. The efficacy of the citrus fruits is widely recognized.

So much misconception and confusion exist, in the minds of those not specially conversant with the subject, about the effect of heat and culinary or preservative processes on the antiscorbutic vitamine that it is important to consider the latest findings as they are reported. The decrease in antiscorbutic potency that milk experiences on heating under certain conditions has too often been translated without reservation to all antiscorbutic foods and finally to all vitamines. Vitamine C is, in truth, much more readily destroyed than is vitamine B by both oxidation and heating, but the rate of destruction depends on a variety of conditions, including the duration of the heating, the opportunity for oxidative

changes, and the reaction of the medium. These facts are reflected in the statement of the better preservation of "freshness" and antiscorbutic value in cold storage than in storage at ordinary temperature, and the serious loss of this property when food is kept hot for a long time between cooking and serving.

Givens, McClugage and Van Horne have demonstrated anew the antiscorbutic property of raw bananas and apples, two fruits finding widespread favor in many parts of the world. As bananas are almost invariably eaten raw in this country, no significant problem of deterioration of vitamine potency arises in connection with them. This is not equally the case with apples, which are consumed in a considerable variety of conditions. According to the latest evidence, such application of temperature as is ordinarily employed in preservation by desiccation or canning tends to reduce the amount of antiscorbutic vitamine of the original raw apple markedly. This is a practical fact of importance. We must learn not only where vitamines may be found in our common foods but also the conditions under which they are preserved intact.—*Jour. Amer. Med. Assoc.*, May 13, 1922.

COMPLIMENTS FOR OUR MAY JOURNAL

[Herewith are published excerpts from a few of the many congratulatory letters and telegrams received at our office. We wish to thank our many readers for these appreciative messages.]

Congratulations on the May number of the *Journal*. It is very attractive and I am sure will do good. The California boys must surely appreciate your efforts to make their meeting a success. No meeting has been boomed like this one, and I hope it will bear fruit. You have done yourself proud in this May issue, and I am sure everybody will be pleased. The printer should also be congratulated on this number.

(Signed) C. N. JOHNSON

May *Journal* excellent. Accept congratulations.

(Signed) C. M. BENBROOK

I wish to compliment you on the fine issue of the May *Journal*. It should prove a good boost for the Los Angeles meeting.

(Signed) CHARLES E. RICE

I wish to thank you personally for the tremendous effort that you put forth in order to bring out such a wonderful number. I know it will not only stimulate the members of our arrangement committee but all of the men in Southern California to put forth an extra effort to make our meeting here a success.

(Signed) J. F. COOK

Accept my sincere congratulations on the May issue of the *Journal*. Without a doubt it is the finest publication we have ever had. May our Convention be likewise.

(Signed) W. E. SIBLEY

May *Journal* splendid. Accept congratulations.

(Signed) BERT BOYD

PROGRAM OF GENERAL SESSIONS

TO BE HELD IN

LOS ANGELES, CALIFORNIA, JULY 17, 18, 19, 20, 21, 1922

TUESDAY MORNING, JULY 18

9:45 A. M.

FIRST GENERAL SESSION

(Theater, Hotel Ambassador)

Invocation

By Rev. M. Howard Fagan

Address of Welcome

By Rufus Bernhard von Kleinsmid, president University of Southern California, on behalf of educational institutions of California.

By Sylvester L. Weaver, ex-president Los Angeles Chamber of Commerce, on behalf of City of Los Angeles.

By Charles M. Benbrook, on behalf of dental profession of California.

Response to Address of Welcome

By Thomas P. Hinman.....Atlanta, Ga.

President's Address

By Thomas B. Hartzell.....Minneapolis, Minn.

TUESDAY EVENING, JULY 18

8:00 P. M.

SECOND GENERAL SESSION

(Theater, Ambassador Hotel)

(In charge of the officers of the section on Histology, Physiology, Pathology, Bacteriology and Chemistry [Research], Weston A. Price, Chairman.)

HEALTH CONSERVATION SYMPOSIUM

1. What rules for diet, in the interest of developing teeth, may be considered safest in the light of present knowledge.
   By Wallace Secombe.....Toronto, Ontario
2. What rules should, in general, govern for the prevention of dental caries and gingival lesions.
   By Russell H. Bunting and U. G. Rickert.....Ann Arbor, Mich.
3. What rules should, in general, govern in deciding what teeth should and what should not be root filled.
   By Clarence J. Grieves.....Baltimore, Md.
4. What rules should, in general, govern for determining whether relatively normal teeth may be sacrificed and definitely abnormal teeth retained for the best interest of the patient.
   By Henry E. Friesell.....Pittsburgh, Pa.
5. What rules should, in general, govern when deciding what methods should be employed for restoring missing teeth.
   By Thomas P. Hinman.....Atlanta, Ga.
6. What rules should, in general, govern in making dental diagnosis.
   By Weston A. Price.....Cleveland, Ohio

WEDNESDAY EVENING, JULY 19

7:45 TO 10:30 P. M.

THIRD GENERAL SESSION

(Trinity Auditorium, 851 S. Grand Ave.)

(In charge of the section on Mouth Hygiene and Preventive Dentistry, Justin D. Towner, chairman.)

Musical Program

"Dental Health Work in Massachusetts"

By Evelyn C. Schmidt, mouth hygiene instructor of the Massachusetts Department of Public Health.....Lynn, Mass.

"Fundamental Problems in Preventive Dentistry"

By Guy S. Millberry....San Francisco, Cal.

Synopsis—A brief presentation of the findings of dental surveys as an introduction to the subject. The etiology of this civic problem, analyzing it as a community, or national disease. The methods proposed and adopted for its treatment and prevention. The economic possibilities in terms of dollars and sense.

"Ten Years to Life"

By Thomas B. Hartzell.....Minneapolis, Minn.

OFFICIAL CALL

The House of Delegates will convene at 10:30 A. M., Monday, July 17, at the Ambassador Hotel.

In the House[1] the representation of the various constituent societies for 1922 is as follows:[2]

Alabama	90
Alaska	
Arizona	42
Arkansas	132
Army Dental Corps	72
Southern California	671
California State	821
Colorado	338
Connecticut	436
Cuba	1
Delaware	33
District of Columbia	215
Florida	45
Foreign	5
Georgia	357
Hawaiian Islands	17
Idaho	56
Illinois	2753
Indiana	681
Iowa	972
Kansas	332
Kentucky	311
Louisiana	236
Maine	51
Maryland	268
Massachusetts	1231
Michigan	1107
Minnesota	1130
Mississippi	194
Missouri	993
Montana	72
National Capitol	13
Navy Dental Corps	31
Nebraska	418
Nevada	44
New Hampshire	46
New Jersey	866
New Mexico	59
New York	2463
North Carolina	295
North Dakota	142
Ohio	1635
Oklahoma	314
Oregon	236
Pennsylvania	1970
Porto Rico	47
U. S. Public Health Service	42
Philippine Islands	23
Rhode Island	
South Carolina	100
South Dakota	182
Tennessee	272
Texas	592
Trustees[3]	
Utah	86
Vermont	84
Virginia	308
Washington	455
West Virginia	261
Wisconsin	996
Wyoming	47

[1] "Sec. 2.—The total voting membership in the House of Delegates, exclusive of the Board of Trustees, shall be as follows: One delegate shall be allowed from each Scientific Section, from the Army Dental Corps, from the Navy Dental Corps, and one from each Constituent Society. Each Constituent Society shall be entitled to one additional delegate if one hundred of its members become regularly enrolled as members of the National Dental Association; and one more delegate for each additional two hundred members enrolled.

Explanatory.—Regardless of enrollment in the National Dental Association each constituent society would have one delegate.

With 100 members in the National, two delegates.

With 300 members in the National, three delegates.

With 500 members in the National, four delegates.

With 700 members in the National, five delegates.

With 900 members in the National, six delegates.

With 1100 members in the National, seven delegates.

With 1300 members in the National, eight delegates.

With 1500 members in the National, nine delegates."—Constitution and By-laws, Art. V, Sec. 2.

[2] This roll will be officially revised June 17, 1922.

[3] "The Trustees shall be members of the House of Delegates without the right to vote."—Constitution and By-laws, Article V, Section 1.

MEMBERS OF THE HOUSE OF DELEGATES, 1922

A Preliminary Roster of the Legislative Body of the National Dental Association

DELEGATES AND ALTERNATES, 1922

This list of members of the House of Delegates for the session is incomplete, as a few of the state societies are yet to report election of their delegates.

ALABAMA

Delegates

Alternates

ALASKA

Delegates

Charles E. Daggett, Cordova

Alternates

Louis S. Keller, Skagway

ARIZONA

Delegates

H. H. Braxton, Phoenix

J. A. Lentz, Phoenix

Alternates

J. C. Roberts, Phoenix

A. J. Mackey, Flagstaff

ARKANSAS

Delegates

J. D. Jordan, 510 Boyle Bldg., Little Rock

G. C. Jernigan, Rector

Alternates

F. G. White, First National Bank Bldg., Fort Smith

J. W. Deaton, Stamps

ARMY DENTAL CORPS

Delegates

Alternates

CALIFORNIA

Delegates

R. B. Giffen, Hagelstein Bldg., Sacramento

Guy S. Millberry, First and Parnassus Aves., San Francisco

John F. McMath, Thomson Bldg., Oakland

D. H. Leppo Union Trust and Savings Bank Bldg., Santa Rosa

Edward J. Howard, 350 Post St., San Francisco

Alternates

A. V. Acker, 302 Bank of Italy, Fresno

F. A. McCan, Elk's Bldg., Stockton

C. H. Henderson, Brown Bldg., Martinez

Howard B. Kirtland, San Luis Obispo

Charles H. Parker, 88 Pacific Ave., Santa Cruz

SOUTHERN CALIFORNIA

Delegates

Charles M. Benbrook, 707 Auditorium Ag., Los Angeles

J. Franklyn Cook, 1001 Merchants National Bank Bldg., Los Angeles

Wallace H. Spinks, 910 Security Bldg., Los Angeles

Grover C. Todd, Hollinsworth Bldg., Los Angeles.

E. H. Lyman, San Marcos Bldg., Santa Barbara

Alternates

F. B. Damron, 608 First National Bank Bldg., Long Beach

A. V. Allen, 445 Chamber of Commerce Bldg., Pasadena

W. A. Brown, 328 Investment Bldg., Pomona

H. Jerome Allen, San Marcos Bldg., Santa Barbara

A. H. Gilbert, Gilbert Blk., San Diego

COLORADO

Delegates

W. T. Chambers, 719 Metropolitan Bldg., Denver

C. S. Kramer, M. W. A. Sanitarium, Woodmen

H. W. Wilson, Metropolitan Bldg., Denver

Alternates

E. E. Bailey, 316 Metropolitan Bldg., Denver

J. W. Needles, 611 Thatcher Blk., Pueblo

J. W. Seybold, 304 Mack Blk., Denver

CONNECTICUT

Delegates

John F. Barton, 197 Asylum Ave., Hartford

Edward S. Gaylord, 63 Trumbull St., New Haven

J. B. Hertz, 194 Atlantic St., Stamford

Alternates

Albert W. Crosby, 29 College St., New Haven

F. J. Erbe, 111 W. Main St., Waterbury

Alfred C. Fones, 10 Washington Ave., Bridgeport

DELAWARE

Delegates

J. D. Brown, Tenth nad Madison Sts., Wilmington

Alternates

H. M. Hogue

DISTRICT OF COLUMBIA

Delegates

Fenton Bradford, The Rochambeau, Washington

Mark F. Finley, 1928 I St., N.W., Washington

Alternates

A. J. Brown, Riggs Bldg., Washington

Thomas L. Rust, 1410 L St., N.W., Washington

FLORIDA

Delegates

C. L. Nance, First National Bank Bldg., Tampa

Alternates

GEORGIA

Delegates

Alternates

HAWAIIAN ISLANDS

Delegates

Clifford B. High, Honolulu

Alternates

IDAHO

Delegates

Alternates

ILLINOIS

Delegates

E. F. Hazell, 608 E. Capitol Ave., Springfield
J. H. Prothero, 22 E. Washington St., Chicago
Arthur G. Smith, 511 Central National Bank Bldg., Peoria
Charles R. Baker, 627 Grove St., Evanston
T. W. Brophy, 81 E. Madison St., Chicago
E. D. Coolidge, 25 E. Washington St., Chicago
G. W. Dittmar, 59 E. Madison St., Chicago
V. H. Fuqua, 25 E. Washington St., Chicago
C. N. Johnson, 22 E. Washington St., Chicago
G. G. Knapp, 1203 W. Garfield Blvd., Chicago
Josephine Pfeifer, 25 E. Washington St., Chicago
H. B. Pinney, 108 N. State St., Chicago
M. M. Printz, 25 E. Washington St., Chicago
P. G. Puterbaugh, 29 E. Madison St., Chicago
M. L. Hanaford, 309 Stewart Office Bldg., Rockford
C. L. Snyder, 505 Second National Bank Bldg., Freeport
W. F. Whalen, 906 Lehman Bldg., Peoria

Alternates

E. C. Pendleton, Golden
L. L. Davis, 81 E. Madison St., Chicago
L. L. Rice, Bridgeport
Hugo Fisher, 25 E. Washington St., Chicago
H. A. Frankel, 190 N. State St., Chicago
F. W. Gethro, 122 S. Michigan Ave., Chicago
A. F. James, 25 E. Washington St., Chicago
R. L. Lewis, 25 E. Washington St., Chicago
H. F. Methven, 25 E. Washington St., Chicago
W. T. Reeves, 159 N. State St., Chicago
F. B. Rhobotham, 25 E. Washington St., Chicago
F. E. Roach, 59 E. Madison St., Chicago
C. F. B. Stowell, 25 E. Washington St., Chicago
H. J. Tharp, 11206 S. Michigan Ave., Chicago
G. N. West, 32 N. State St., Chicago
A. C. Willman, Cobb Bldg., Kankakee
L. H. Wolfe, Quincy

INDIANA

Delegates

C. R. Jackson, 907 Hume-Mansur Bldg., Indianapolis
A. R. Ross, Lafayette
P. H. Chadwick, Rushville
W. E. Neuenschwander, 2015 Broadway, Ft. Wayne
F. R. Henshaw, 507 Pythian Bldg., Indianapolis
H. C. McKittrick, 1006 I. O. O. F. Bldg., Indianapolis
L. W. Dailey, Bluffton

Alternates

S. F. Gilmore, 1002 I. O. O. F. Bldg., Indianapolis
G. E. Bell, Lafayette
F. R. McClanahan, Rushville
Harry S. Shields, Brownstown
E. R. Kibler, 1006 Hume-Mansur Bldg., Indianapolis
W. O. Vallette, Goshen
A. B. Price, 14 Colonial Bldg., Richmond

IOWA

Delegates

Earle S. Smith, 635 S. Governor St., Iowa City
L. W. Snuggins, Des Moines
Fred Waters, Ames
C. R. Baker, Davenport
F. T. Breene, Iowa City
J. V. Conzett, Dubuque
S. M. Work, Ottumwa

Alternates

M. B. Loomis, Cedar Rapids
Erling Thoen, Iowa City

KANSAS

Delegates

G. A. Crise, Manhattan
Geo. E. Burkett, Kingman
F. O. Hetrick, Ottawa
H. M. McFarland, Wohleman Bldg., Kansas City

Alternates

W. T. Triplett, Salina
W. A. McKee, Hiawatha
W. A. Coston, 100 Kansas Ave., Topeka
C. K. Weaver, Clay Center

KENTUCKY

Delegates

I. H. Harrington, 340 Frances Bldg., Louisville
W. F. Walz, 159 E. Main St., Lexington
H. B. Tileston, 916 Starks Bldg., Louisville

Alternates

G. H. Greer, 2704 Portland Ave., Louisville
W. W. Wilkinson, Paducah
W. A. Bridwell, Providence

LOUISIANA

Delegates

J. P. Wahl, 1135 Maison-Blanche Bldg., New Orleans
R. D. Tilley, Commercial Bank Bldg., Shreveport

Alternates

S. S. Grosjean, 816 Howard Ave., New Orleans
T. M. Terry, Godchaux Bldg., New Orleans

MARYLAND

Delegates

B. Lucien Brun, 827 Park Ave., Baltimore
Harry E. Kelsey 817 Park Ave., Baltimore

Alternates

James H. Ferguson, Jr., "The Homewood" 914 N. Charles St., Baltimore
A. M. Russell, University of Maryland Dental Department, Baltimore

MASSACHUSETTS

Delegates

Leon A. Storz, 507 Main St., Worcester
Frederick W. Allen, 520 Beacon St., Boston
A. W. Doubleday, 220 Marlboro St., Boston
W. H. Eaton, 419 Boylston St., Boston
T. P. Ernest Greene, 342 Union St., New Bedford
Percy Howe, 199 Marlboro St., Boston
Carl R. Lindstrom, 520 Beacon St., Boston
Murdoch C. Smith, 3 Lee Hall, Lynn

Alternates

Frank H. Dean, 311 Main St., Worcester
W. H. Gilpatric, 358 Commonwealth Ave., Boston
F. E. Jeffrey, 219 Essex St., Salem
Charles M. Proctor, 19 Bay State Road, Boston
W. H. Richardson, 390 Main St., Worcester
William J. Speers, 78 Bedford St., Fall River
David F. Spinney, 1330 Beacon St., Brookline
F. J. Sullivan, 124 Franklin St., Fall River

MICHIGAN

Delegates

Bion R. East, 62 W. Adams St., Detroit
W. A. Giffen, 609 Washington Arcade, Detroit
A. L. LeGro, 525 David Whitney Bldg., Detroit
C. G. Bates, Durand
C. G. Parker, 1047 David Whitney Bldg., Detroit
W. H. Elliott, 7643 Jefferson Ave., E., Detroit

Alternates

B. S. Sutherland, Owosso
C. J. Lyons, Ann Arbor
E. J. Anderson, Manistee
J. J. Travis, 721 University Ave., Ann Arbor
W. W. Gibson, 201 Loraine Bldg., Grand Rapids
P. R. Glass, Saginaw

MINNESOTA

Delegates

Max E. Ernst, 1150 Lowry Bldg., St. Paul

O. DeForest Davis, 933 Metropolitan Bank Bldg., Minneapolis

H. B. Washburn, Raymond and University, St. Paul

C. E. Conley, Le Sueur

Benjamin Sandy, 636 Syndicate Bldg., Minneapolis

T. W. Maves, 500 Donaldson Bldg., Minneapolis

C. C. Sparrow, 624 La Salle Bldg., Minneapolis

Alternates

W. V. Buck, 1114 Lowry Bldg., St. Paul

A. F. Englund, 408 Donaldson Bldg., Minneapolis

George M. Damon, 730 La Salle Bldg., Minneapolis

C. H. Beers, 1690 Grand Ave., St. Paul

W. D. Vehe, 1010 Donaldson Bldg., Minneapolis

E. T. Tinker, 401 Donaldson Bldg., Minneapolis

W. D. Card, 800 La Salle Bldg., Minneapolis

MISSISSIPPI

Delegates

J. F. Brunson, Meridian

W. R. Wright, Jackson

Alternates

J. R. Moore, Greenwood

N. L. Swayze, Yazoo City

MISSOURI

Delegates

W. A. Roddy, Frisco Bldg., St. Louis

R. J. Rinehart, Bryant Bldg., Kansas City

George E. Haigh, Jefferson City

J. P. Harper, 3564 Caroline St., St. Louis

J. D. Patterson, Keith and Perry Bldg., Kansas City

E. P. Brady, 5238 Menerva Ave., St. Louis

H. E. Allee, Clarksburg

Frank C. Rodgers, Wall Bldg., St. Louis

Alternates

Jesse W. Miller, Jr., Maryville

Z. W. Street, Sturgeon

T. H. Tye, Cameron

G. T. Reily, Fulton

Charles W. Digges, Columbia

Thomas J. Sugg, Carrollton

A. J. Thompson, Shukert Bldg., Kansas City

J. W. Hull, Commerce Bldg., Kansas City

MONTANA

Delegates

Alternates

NATIONAL CAPITAL

Delegates

Alternates

NAVY DENTAL CORPS

Delegates

Lieutenant Commander William N. Cogan, Navy Yard, Mare Island, California

Alternates

Lieutenant Commander James L. Brown, U.S. SS. Mercy, Care Postmaster, San Francisco, Cal.

NEBRASKA

Delegates

Alternates

NEVADA

Delegates

Alternates

NEW HAMPSHIRE

Delegates

Ouida Mackey Gorham, 15 N. Main St., Concord

Alternates

NEW JERSEY

Delegates

Alternates

NEW MEXICO

Delegates

C. A. Eller, Albuquerque

H. S. Murdock, Springer

Alternates

C. W. Hoover, Dawson

C. H. Woolgar, Magdalena

NEW YORK

Delegates

Alternates

NORTH CAROLINA

Delegates

Alternates

NORTH DAKOTA

Delegates

Albert Hallenberg, Fargo

William E. Hocking, Devils Lake

Alternates

James W. Hamilton, Minot

William J. Hewitt, Grand Forks

OHIO

Delegates

H. C. Brown, 609 Hartman Bldg., Columbus

J. F. Stephan, 1402 Guardian Bldg., Cleveland

E. C. Mills, 151 E. Broad St., Columbus

F. M. Casto, 458 Rose Bldg., Cleveland

L. L. Barber, 718 Spitzer Bldg., Toledo

C. W. Mills, Chillicothe

C. H. Schott, 1004 Neave Bldg., Cincinnati

W. A. Price, 8926 Euclid Ave., Cleveland

Harry Cope, 328 E. State St., Columbus

Alternates

W. H. Hayden, 228 Lincoln Ave., Youngstown

G. D. Edgar, Defiance

Z. N. Wright, 730 Fidelity Bldg., Dayton

E. S. Braithwaite, Willard

H. M. Semans, 710 Park St., Columbus

J. K. Douglas, Sandusky

J. J. Welker, 1143 Ohio Bldg., Toledo

E. L. Pettibone, 6503 Detroit Ave., Cleveland

T. I. Way, 52 Groton Bldg., Cincinnati

OKLAHOMA

Delegates

C. W. Day, Temple Apts., Tulsa

C. R. Lawrence, Enid

C. W. White, Colcord Bldg., Oklahoma City

Alternates

OREGON

Delegates

W. C. Shearer, Stevens Bldg., Portland

A. W. Chance, 810 Corbett Bldg., Portland

T. Rex Baldwin, Stevens Bldg., Portland

Alternates

H. C. Fixott, 553 Morgan Bldg., Portland

Jean Cline, Broadway Bldg., Portland

W. Claude Adams, 1203 Selling Bldg., Portland

PENNSYLVANIA

Delegates

H. E. Friesell, 1206 Highland Bldg., Pittsburgh
E. R. Sausser, 225 S. Fifteenth St., Philadelphia
J. F. Biddle, 517 Arch St., Pittsburgh
R. H. D. Swing, 1623 Walnut St., Philadelphia
W. L. Fickes, East End Savings and Trust Co., Pittsburgh
O. J. Specker, 51 N. Fourth Ave., Reading
A. P. Lee, 1616 Locust St., Philadelphia
A. C. Young, Highland Bldg., Pittsburgh
T. W. Brand, 7124 Jenkins Arcade, Pittsburgh
F. C. Friesell, 1206 Highland Bldg., Pittsburgh
Leslie Waddill, Jenkins Arcade, Pittsburgh
Robert Zugsmith, Jenkins Arcade, Pittsburgh

Alternates

A. C. Barclay, 702 Highland Bldg., Pittsburgh
L. E. Van Kirk, 1123 Pennsylvania Ave., Pittsburgh
J. C. G. Fitzhugh, Masonic Temple Bldg., McKeesport
Emmett O'Neil, 811 Medical Arts Bldg., Philadelphia
W. C. Puhl, Real Estate Savings and Trust Bldg., Pittsburgh
W. E. Mendell, 405 E. Ohio St., Pittsburgh
J. B. Duffield, Locust and Fifteenth Sts., Philadelphia
A. H. Reynolds, 4630 Chester Ave., Philadelphia
C. S. Harris, 7023 Jenkins Arcade, Pittsburgh
A. L. Barelitt, Highland Bldg., Pittsburgh
T. B. Magee, Ellwood City
J. H. Logan, Portage

PHILIPPINE ISLANDS

Delegates

Alternates

PORTO RICO

Delegates

Alternates

RHODE ISLAND

Delegates

Alternates

SOUTH CAROLINA

Delegates

Alternates

SOUTH DAKOTA

Delegates

J. H. Martin, Vermilion
E. D. Buettell, Mitchell

Alternates

H. G. Tarbell, Watertown
P. A. Eernisse, Minnekahta Blk., Hot Springs

TENNESSEE

Delegates

Alternates

TEXAS

Delegates

J. O. Hall, Amicable Bldg., Waco
A. L. Frew, 736 Wilson Bldg., Dallas
J. M. Murphy, Temple
C. W. Smith, 501 S. W. Life Insurance Bldg., Dallas

Alternates

Allen N. Kearby, Dallas County Bank Bldg., Dallas
W. H. Scherer, Scanlan Bldg., Houston
G. Waller Staples, Wilson Bldg., Dallas
Trim Houston, Corsicana

TRUSTEES

R. H. Volland, Iowa City, Iowa
C. C. Allen, Tenth and Troost, Kansas City, Mo.
John D. Millikin, Elkan Gunst Bldg., San Francisco, Cal.
Thomas P. Hinman, Fourth National Bank Bldg., Atlanta, Ga.
H. J. Burkhart, 300 Sibley Blk., Rochester, N. Y.
C. W. Hall, Alhambra Bldg., Milwaukee, Wis.
Waldo E. Boardman, 419 Boylston St., Boston, Mass.
Clarence J. Grieves, 201 Madison St., Baltimore, Md.
Carl D. Lucas, 902 Hume-Mansur Bldg., Indianapolis, Ind.

UTAH

Delegates

W. A. Squires, Boston Bldg., Salt Lake
R. W. Hall, Kearns Bldg., Salt Lake

Alternates

Victor H. Sears, Clift Bldg., Salt Lake
Leo J. Shepard, Boston Bldg., Salt Lake

VERMONT

Delegates

George M. Somers, Proctor

Alternate

George D. Samson, Burlington

VIRGINIA

Delegates

J. A. C. Hoggan, 506 Professional Bldg., Richmond
Harry Bear, 410 Professional Bldg., Richmond

Alternates

WASHINGTON

Delegates

J. H. Winstanley, Waterville
R. S. Williams, National Realty Bldg., Tacoma
F. H. Hergert, Cobb Bldg., Seattle

Alternates

WEST VIRGINIA

Delegates

H. W. Burnett, Fairmont
H. T. Halstead, 637 Thirteenth Ave., Huntington
S. T. Bird, Princeton

Alternates

R. Earle McCray, Fairmont
S. M. Callaway, Huntington
J. C. Houchins, North Fork

WISCONSIN

Delegates

W. H. Cudworth, 128 Wisconsin St., Milwaukee
H. L. Banzhaf, 401 First National Bank Bldg., Milwaukee
A. G. Fee, Superior
T. A. Hardgrove, Fond du Lac
J. L. Blish, Fond du Lac
J. J. Wright, 1218 Wells Bldg., Milwaukee

Alternates

D. P. Wheeler, Madison
E. M. Kapitan, Manitowoc
E. A. Geilfuss, 809 Goldsmith Bldg., Milwaukee
E. J. Weaver, 505 Realty Exchange Bldg., Milwaukee

WYOMING

Delegates

I. P. Hayes, Sheridan

Alternates

C. H. Bailey, Midwest Bldg., Casper

Announcements

CALENDAR OF MEETINGS

NATIONAL SOCIETIES

National Dental Association, July 17-21, 1922, Ambassador Hotel, Los Angeles, Cal. Otto U. King, Secretary, 5 N. Wabash Ave., Chicago, Ill.

National Society of Denture Prosthetists, July 3-15, Dental Department, University of Southern California, Los Angeles, Cal. E. B. Owen, Secretary, 335 Frisco Bldg., St. Louis, Mo.

National Association of Dental Examiners, July 17 and 18, Ambassador Hotel, Los Angeles, Cal. H. C. McKittrick, Secretary, 1006 I. O. O. F. Bldg., Indianapolis, Ind.

National Association of Dental Faculties, July 13, 14, 15, Hotel Clark, Los Angeles, Cal. Herbert L. Wheeler, Secretary, 560 Fifth Ave., New York, N. Y.

National Dental Sorority, July 17, Ambassador Hotel, Los Angeles, Cal. Mae Fontaine, Secretary, Consolidated Realty Bldg., Los Angeles, Cal.

American Dental Library and Museum Association, July 17, Ambassador Hotel, Los Angeles, Cal. B. W. Weinberger, Secretary, 42 E. 41st St., New York, N. Y.

American Society of Oral Surgeons and Exodontists, July 11-14, Hotel Alexandria, Los Angeles, Cal. Joseph P. Wahl, Secretary, 1135 Maison-Blanche Bldg., New Orleans, La.

American Society of Dental Radiographers, July 19 and 20, Ambassador Hotel, Los Angeles, Cal. Martin Dewey, Secretary, 501 Fifth Ave., New York, N. Y.

Association of Military Dental Surgeons, July 18, Hotel Ambassador, Los Angeles, Cal. Rea Proctor McGee, President, 613 Jenkins Bldg., Pittsburgh, Pa.

American Academy of Periodontology, July 10, 11, 1922, Drake Hotel, Chicago, Ill. J. Herbert Wood, Secretary, Cleveland, Ohio.

American Dental Society of Europe, July 28-31, London, England. William A. Spring, President, 8 W. 40th St., New York, N. Y. F. W. Fleming, Secretary, 13 Queen Anne St., London.

American Public Health Association, October 16-19, Cleveland, Ohio.

Interstate Activities (meeting of delegates) July 19, Hotel Ambassador, Los Angeles, Cal. John E. Gurley, Chairman, 350 Post St., San Francisco, Cal.

Pacific Coast Society of Orthodontists, July 13, 14, 15, Hotel Angelus, Los Angeles, Cal. Carl O. Engstrum, Secretary, 306 Hagelstein Bldg., Sacramento, Cal.

Delta Sigma Delta Alumni and Grand Chapter Fraternity, July 17, Hotel Alexandria, Los Angeles, Cal. R. Hamel D. Swing, Supreme Scribe, 1623 Walnut St., Philadelphia, Pa.

Psi Omega Alumni Fraternity, July 17, Hotel Clark, Los Angeles, Cal. Max Wassman, Jr., Grand Master, 409 Elkan-Gunst Bldg., San Francisco, Cal.

Xi Psi Phi Alumni Fraternity, July 17, Hotel Hayward, Los Angeles, Cal. J. Emmett Northcutt, Secretary, St. John and Elmwood Ave., Kansas City, Mo.

STATE SOCIETIES

June

California (Southern), at Los Angeles.
Colorado, at Colorado Springs (15, 16, 17).
Georgia, at Atlanta (14, 15, 16).
Maine, at Augusta (21, 22, 23).
New Hampshire, at The Wiers (26, 27, 28).
New Mexico, at Albuquerque (26, 27, 28).
North Carolina, at Wrightsville Beach (12, 13, 14).
South Carolina, at Georgetown (13, 14, 15).
Utah, at Salt Lake City (19, 24).
Wisconsin, at Milwaukee (27, 28, 29).
Wyoming, at Sheridan (20, 21, 22).

July

Montana, at Helena, (6, 7, 8).
Utah, at Los Angeles, Cal. (19).

August

Washington, at Seattle (2-5).

September

Delaware, at Wilmington (14).

October

Northern Indiana, at Wabash (2, 3).
Virginia, at Richmond (18-21).

MEETINGS OF STATE BOARDS OF DENTAL EXAMINERS

Alabama, at Birmingham Dental College, Birmingham, June 19. Secretary, H. Clay Hassell, 616 Twenty-second Ave., Tuscaloosa.

Arkansas, at Hotel Marion, Little Rock, June 19-21. Secretary, H. J. Crume, Wilson Bldg., El Dorado.

Connecticut, at Hartford, June 22-24. Recorder, Robert H. W. Strang, 886 Main St, Bridgeport.

Colorado, at Denver, June 6. Secretary, William H. Flint, Littleton.

Delaware, at Municipal Building, Tenth and King Streets, Wilmington, July 12 and 13. Secretary, W. S. P. Combs, P. O. Box 221, Middletown.

Florida, at Jacksonville, June 7, 8, 9, 10. Secretary, R. P. Taylor, Jacksonville.

Georgia, at Capitol, Atlanta, May 29 and June 14. Secretary D. D. Atkinson, Brunswick.

Idaho, at Boise, Bureau of License, January 10 and July 11, 1922. Paul Davis, Director, Bureaus of License, Boise.

Indiana, at Indianapolis, June 12-17. H. C. McKittrick, Secretary, 1006 I. O. O. F. Building, Indianapolis.

Kansas, June 19-23, at Wichita, G. F. Ambrose, Secretary, El Dorado.

Maine, at State House, Augusta, July 6, 7, and 8. Secretary, Henry Gilman, Portland.

Maryland, at University of Maryland, Baltimore, June 7, 8 and 9. Secretary, F. F. Drew, 701 N. Howard St., Baltimore.

Massachusetts, at Boston, June 26, 27, 28, 29, 30, and July 1. Secretary, J. N. Carriere, 146 State House, Boston.

Michigan, at Dental College, Ann Arbor, June 5-11. Secretary, E. O. Gillespie, 745 David Whitney Building, Detroit.

Minnesota, at College of Dentistry, University of Minnesota, June 15. Secretary, C. G. Gillam, Babcock Bldg., Austin.

Mississippi, Senate Chamber, State Capitol Building, Jackson, June 20. B. J. Marshall, Secretary, Marks.

Missouri, at St. Louis University, St. Louis, June 9-13. George E. Haigh, Secretary, Jefferson City.

Montana, at Helena, July 10. Secretary, Frank J. Bell, Billings.

Nebraska, at Creighton Dental College, Omaha, and University of Nebraska Dental College, Lincoln, and at the State House, Lincoln, June 6-10. Secretary, H. H. Antles, Department of Public Welfare, Lincoln.

New Jersey, at Trenton, June 26-30. Secretary, John C. Forsyth, 429 E. State St., Trenton.

North Carolina, at Wrightsville Beach, Wilmington, June 8. Secretary, F. L. Hunt, Asheville.

North Dakota, at Fargo, July 11. Secretary, W. E. Hocking, Devils Lake.

Oklahoma, at Oklahoma City, June 26. L. M. Doss, Secretary, American National Bank Building, Oklahoma City.

Pennsylvania, at Pittsburgh and Philadelphia, June 21-24. Secretary, Alexander H. Reynolds, 4630 Chester Ave., Philadelphia, Pa.

Rhode Island, at State House, Providence, July 5, 6, and 7. Secretary, Albert L. Midgley, 315 Butler Exchange, Providence.

South Carolina, at Jefferson Hotel, Columbia, June 9. Secretary, W. K. Walker, Orangeburg.

South Dakota, at Sioux Falls, June 26. Secretary, L. R. Walston, Redfield.

Tennessee, at Vanderbilt University Dental Department, Nashville, June 19. Secretary, F. W. Meacham, 911 Hamilton National Bank Bldg., Chattanooga.

Texas, at Dallas, June 19-22. R. L. Rogers Secretary, Amarillo.

Vermont, at State House, Montpelier, June 26, 27, 28. Secretary, Harry F. Hamilton, Newport.

Virginia, at Richmond, June 13. Secretary, J. P. Stiff, Fredericksburg.

West Virginia, at Wheeling, June 27. Secretary, R. Mason Hite, Mannington.

Wisconsin, June 19-24. J. L. Blish, Secretary, Fond du Lac.

MONTANA STATE DENTAL SOCIETY

The ninteenth annual meeting will be held in Helena, July 6, 7 and 8. A cordial invitation is extended to all members of recognized dental societies.

J. E. BAKER, *Secretary-Treasurer,*
311 Ford Bldg., Great Falls, Mont.

NEW MEXICO STATE DENTAL SOCIETY

The New Mexico State Dental Society will hold its annual meeting at Albuquerque, June 26, 27 and 28. An interesting program is being arranged. All ethical members of the profession are invited to attend.

D. P. NOLTING, *Secretary,*
Albuquerque, N. M.

DELAWARE BOARD OF DENTAL EXAMINERS

The next meeting of the Delaware Board of Dental Examiners will be held July 12 and 13, Municipal Building, Tenth and King Streets, Wilmington.

For further information apply to

W. S. P. COMBS, *Secretary,*
Middletown, Del.

THE AMERICAN DENTAL SOCIETY OF EUROPE

The meeting of the American Dental Society of Europe will be held at the Hotel Cecil, the Strand, and the hotel has offered a limited number of rooms to members and guests at the following rates: single room, with attendance, use of bathroom and table d'hote breakfast, fifteen shillings per day; double room inclusive above for two persons, twenty-seven shillings per day; single room with private bath and lavatory attached, attendance and table d'hote breakfast, twenty-four shillings per day; double room with private bath and lavatory attached, attendance and table d'hote breakfast for two persons, thirty-seven shilling per day.

Members of the profession all over the world are cordially invited.

WILLIAM A. SPRING, *President*,
8 W. 40th St., New York, N. Y.
F. W. FLEMING, *Secretary*,
13 Queen St., London, Eng.

MASSACHUSETTS STATE DENTAL HYGIENISTS' ASSOCIATION

On April 3, 1922, the first meeting of the Massachusetts State Dental Hygienists' Association was held at the Forsyth Dental Infirmary, Boston, Massachusetts.

At this meeting Dr. Frank P. Barnard, of Worcester, acting in behalf of the Massachusetts Dental Society, of which organization he is president, formally recognized the Massachusetts State Dental Hygienists' association, and extended an invitation to the hygienists to hold their future annual meetings in conjunction with the Massachusetts Dental Society. The invitation was gratefully accepted and in the future the two organizations will meet together.

The following officers were elected: president, Mary G. Jackson; vice-president, Freda P. Ward; secretary, Louise Borchardt; treasurer, Cassie Kartzmark; executive committee, M. Dorothy Sprague, Harriet Putman, Bessie MacFarland; editor, Ferna Kendall.

FERNA KENDALL, *Editor*.

XI PSI PHI ALUMNI

The Xi Psi Phi alumni of Pennsylvania, New York, New Jersey and Delaware are asked to kindly communicate with any of the following: William C. T. Bauerle, Zeta Chapter, Flanders Building; John Burkhardt, Pi Chapter, 842 N. Twenty-fourth St.; Philip H. Wood, Gamma Chapter, 4939 Chestnut St., all of Philadelphia. Give name of college, chapter and date of graduation.

WILLIAM C. T. BAUERLE,
Flanders Bldg., Fifteenth and Walnut Sts.,
Philadelphia, Pa.

THE HEALTH EXHIBIT

The Mouth Hygiene and Publicity Council, the object of which is to assist in carrying the gospel of clean, healthy mouths to the millions of this country, has adopted the suggestion of our worthy president, Dr. Hartzell, to arrange a great health exhibit, the like of which no profession has attempted heretofore. This exhibit will show the interdependence of medicine and dentistry, and will be designed to convey to all the importance of good health, and to illustrate how it may be possible to add a decade or more to the average span of human life. It will be open to the public as well as to the profession.

Prevention will be the central idea, and it is expected that each exhibit will portray what can be accomplished thru some phase of human activity related to dentistry. A space of 62 x 118 feet has been allotted to the exhibit, in which will be gathered the results of years of untiring efforts on the part of our scientific and teaching forces.

As to the nature of the exhibit, such material as slogans, graphs, blackboards, lantern slides and films, models, posters, charts, photographs, etc., in fact anything that has been successfully used in putting over mouth hygiene campaigns as well as methods and means will form the exhibition.

It would be well for all to inquire into the activity of their own states and get in line with what they are doing toward making the exhibit the greatest exhibit ever held in the line of dental activity.

All exhibits and contributions should be plainly addressed to HEALTH EXHIBIT, National Dental Association. Citizens Truck Co., Los Angeles, Cal.

W. R. WRIGHT, *Chairman*
Mouth Hygiene Publicity Council,
Jackson, Miss.

ASSOCIATION OF DENTAL EXAMINERS, UNITED STATES VETERAN'S BUREAU

There will be a meeting of the Association of Dental Examiners, United States Veteran's Bureau at Los Angeles, July 17 to 21, 1922.

This Association was formed in Ohio, Indiana, and Kentucky and expects to organize nationally at this meeting.

The purpose of the organization is to promote high ideals and good dental service to the disabled ex-service man who is receiving this treatment by education, science, and mutual fellowship; by union of effort of all members thru organization; by co-operation with the United States Veteran's Bureau and medical department thereof; by advocacy of

proper legislation; by co-operation with all other organizations that will better the dental service to the disabled ex-service man.

P. J. AUFDERHEIDE, *President*
9107 Miles Ave., Cleveland, Ohio.

TROWEL FRATERNITY

The Trowel Fraternity headquarters during the week of the National Dental meeting, July 17-21, will be located at the Ambassador Hotel, Los Angeles, California. For information concerning the same communicate with

L. M. BAUGHMAN,
603 Pantages Bldg., Los Angeles, Cal.

PSI OMEGA FRATERNITY

The National Alumni Chapter of the Psi Omega Fraternity will hold its next meeting on Monday, July 17, at the Clark Hotel, Los Angeles, California. The chapter will be called to order at 10:00 A. M. There will also be an afternoon meeting and an informal banquet at 8:00 P. M. at the Los Angeles Athletic Club, at which the Psi Omega ladies will be the guests of the Fraternity. A large attendance is desired. Every Psi Omega should register immediately on arrival in Los Angeles. Headquarters will be maintained during the National meeting at both the Ambassador and Clark hotels.

MAX WASSMAN, JR.
Grandmaster National Alumni Chapter,
419 Elkan Gunst Bldg., San Francisco, Cal.

AN INVITATION FROM THE PORTLAND DISTRICT DENTAL SOCIETY

Will dentists who expect to travel to the Los Angeles meeting via Portland, please refer to p. A-54 in this issue.

DENTAL CORPS ORDERS FROM WAR DEPARTMENT

NAVY

Lieutenant C. B. Morse, naval prison, Portsmouth, N. H.; to marine expeditionary force, Portau-Prince, Haiti.

Lieutenant Charles H. Morris, naval training station, Hampton Roads, Va.; to receiving barracks, Hampton Roads.—*Army and Navy Register,* March 25, 1922.

Lieutenant J. J. Haas, R. S., Philadelphia; to dispensary, Navy Department.—*Army and Navy Register,* April 1, 1922.

Lieutenant E. C. O'Connell, squadron 10; to squadron 12.—*Army and Navy Register,* April 8, 1922.

Lieutenant C. R. Wells, R. S., Philadelphia, Pa.; to naval hospital, New York.

Lieutenant (J. G.) J. A. Turrentine, Olympia; to marine expeditionary force, Santo Domingo.—*Army and Navy Register,* April 15, 1922.

Lieutenant N. S. Duggan, New Mexico; to Idaho,—*Army and Navy Register,* April 29, 1922.

Lieutenant F. V. Davis, Connecticut; to Eagle 11.

Lieutenant (J. G.) H. J. Hill, Eagle 11; to Connecticut.—*Army and Navy Register,* May 6, 1922.

Lieutenant Commander J. A. Mahoney, naval dispensary, Washington, D. C.; to Henderson.

Lieutenant I. G. Kohlmeier, Naval Academy; to naval dispensary, Navy Department.

Lieutenant (J. G.) H. R. Alexander, Henderson; to naval training station, Hampton Roads, Va.—*Army and Navy Register,* May 13, 1922.

ARMY

Captain George J. Sibley report to retiring board at Presidio of San Francisco for examination.—*Army and Navy Register,* March 25, 1922.

Captain Glover Johns from duty with 5th infantry at Fort Williams, Me., and report for assignment to another station in 1st corps area.

Captain George M. Babbitt upon arrival in United States to Camp Dix, N. J., for duty.—*Army and Navy Register,* April 1, 1922.

Major Hugh M. Tarpley from duty at Fort Hancock, N. J., to New York City and about August 3 to Panama Canal Department for duty.

Major Aaron F. Ridemiller from duties in Panama Canal Zone, effective upon completion of his present tour of foreign service, to duty at Fort Hancock, N. J.

Major Charles C. Mann report to retiring board at Fitzsimons General Hospital, Denver, Colo., for examination.

Captain Joseph H. Jones to William Beaumont General Hospital, El Paso, Tex., for observation and treatment.—*Army and Navy Register,* April 8, 1922.

Captain Daniel J. Crowley from treatment at Walter Reed General Hospital, D. C., and return to his station, Fort Ethan Allen, Vt.—*Army and Navy Register,* April 15, 1922.

Captain Robert E. Motley, now at the U. S. Army and Navy General Hospital, Hot Springs, Ark., will proceed to Fort McPhearson, Ga., and report to Colonel Moses G. Zalinski, quartermaster corps, president of

an Army retiring board at headquarters, 4th corps area, for examination. Upon completion of examination Captain Motley will return to place of receipt of order.—*Army and Navy Register,* April 29, 1922.

Captain Charles L. Andrews report to retiring board at Presidio of San Francisco for examination.

Captain Avery G. Holmes report by telegraph to commanding general, 2d corps area, for assignment to duty and station.

Captain Thomas L. Spoon from duty in office of attending surgeon, Washington, to Camp Bragg, N. C., for duty.

Captain Richard F. Thompson upon arrival in United States to Camp Knox, Ky., for duty.

Captain Harold S. Whitney having been found by an Army retiring board incapacitated for active service on account of disability incident thereto, his retirement from active service is announctd; proceed home.—*Army and Navy Register,* May 13, 1922.

A. A. Dental Surgeon James W. Young of the Public Health Service, relieved from Waukesha, Wis., assigned to Coast Guard cutter Bear.

List of applicants who have accepted appointments in Dental Reserve Corps: First Lieutenant R. F. Berner, Chicago, Ill.; Captain F. F. Harrower, South Norwalk, Conn.; Major A. Owre, Minneapolis, Minn.; Captain L. J. Dunn, Brooklyn, N. Y.; First Lieutenant E. O. Willson, Elgin, Ore.; Captain W. A. Charron, Worcester, Mass.; First Lieutenant L. F. Shade, Berkeley, Cal..

Navy Dental School

A dental school will shortly be established as an adjunct of the Naval Medical School in this city, according to plans of the surgeon general of the Navy. As a preliminary to the establishment of this school, three officers of the dental corps have been selected to undergo a special course of preparation to fit them for duty as instructors. The officers selected are Lieutenant Commander H. E. Harvey, who will have charge of operative work; Lieutenant Commander Wm. L. Darnall, who will be in charge of all surgery, and Lieutenant John J. Haas, who will be in charge of prosthetic work. These officers will shortly proceed to Chicago and take an intensive course in modern dentistry, to be followed by courses in their special line of work. It is expected that Lieutenant Commander Darnall will spend a considerable portion of his time in the large clinics of New York, while Lieutenant Commander Harvey, who will be in charge of operative work, will take a special course of two months in removable bridge work, going first to Chicago and then to New York City. It is not the intention of the dental class to perform laboratory work at the outset, but, as circumstances permit and the personnel become trained and more experienced, it is thought that the naval service will be given the benefit of the most modern dental treatment. The dental school will be a part of the Naval Medical School at 23d and E streets, this city, and will be started about next January. Duplication will be avoided, where the same instruction is given and the same materials used. The three officers designated for instructors will begin their special course of instruction on June 5. A feature of the school will be the eventual training of members of the hospital corps in mechanical dentistry and as assistants to dental officers. —*Army and Navy Register,* May 20, 1922.

Army Dental Corps

Important changes in the Army dental corps are in prospect, due to the transfer from the active list of four or five officers who have been found incapacitated for duty. The papers in these cases are before the general staff, and it is expected that announcement of the retirements will be made shortly, since the War Department has decided that there will be no appreciable delay in the retirements for disability in the case of members of the medical department and the chaplains, as well as first lieutenants, who are on the promotion list. In such cases the retirements create no vacancies to be filled by the promotion of juniors. The dental school, which began its sessions last January at the Walter Reed Hospital, will hold its graduation exercises of the present class in conjunction with the graduation of the Army medical school on June 22. There are 17 student officers, who will be assigned temporary duty at the training camps this summer and later on will be ordered to permanent stations. The next class of dental officers will be assembled about the 1st of September for preliminary instruction at Carlisle Barracks, coming to the institution in Washington in January for the regular six months' course. It is now expected that some 20 officers will be selected for this special instruction next year, but the number actually designated will necessarily depend on the result of pending legislation that affects the commissioned strength of this branch of the medical department. Lieutenant Charles E. Callery, of the corps, who is a student officer of the dental school this year, is undergoing examination for promotion to the grade of captain, for which he is eligible by reason of the required length of service.—*Army and Navy Register,* May 13, 1922.

TRANSPORTATION ARRANGEMENTS FOR LOS ANGELES MEETING

Summer tourist tickets available for this convention will be on sale daily, May 15 to September 30, 1922, inclusive, permitting stop-over at any point en route within final return limit of October 31. These tickets are also on sale to the general public.

Persons buying these tickets *must* map out a complete itinerary on both going and return routes before making the purchase, since no changes will be allowed under and circumstances after tickets are bought.

The following fares to Los Angeles and return have been quoted:

FROM	Direct Routes Going and Returning	One Way via Portland or Seattle
Boston, Mass.	$147 00	$165 66
Baltimore, Md.	130 45	148 45
Buffalo, N. Y. (standard lines)	116 10	134 10
Buffalo, N. Y. (differential lines)	113 70	131 70
Cleveland, Ohio, (standard lines)	105 65	123 65
Cleveland, Ohio, (differential lines)	104 05	122 05
Columbus, Ohio	104 00	122 00
Cincinnati, Ohio	101 35	119 35
Detroit, Mich.	101 70	119 70
Dayton, Ohio	101 40	122 40
Indianapolis, Ind.	95 70	113 70
New York, N. Y. (standard lines)	138 32	156 32
New York, N. Y. (differential lines)	135 12	153 12
Philadelphia, Pa.	133 14	151 14
Pittsburgh, Pa.	113 05	131 05
Toledo, Ohio	99 48	117 48
Chicago, Ill.	86 00	104 00
St. Paul, Minn.	87 50	97 75
Minneapolis, Minn.	87 50	97 75
Des Moines, Iowa	77 65	95 66
Omaha, Nebr.	72 00	90 00
Kansas City, Mo.	72 00	90 00
St. Louis, Mo.	81 50	99 50

Correspondingly low fares will be on sale from all points in the United States and Canada.

For the accommodation of those attending the meeting, the Transportation Committee has completed arrangements for the operation of two official special trains from Chicago, which will afford a choice of routes with a variety of entertainment and scenic points of interest.

You will note from the schedules that both trains will leave Chicago the same evening, arriving the same hour at Colorado Springs, where a most enjoyable day will be spent by the entire party.

The itineraries as outlined, we believe should be very interesting and should help to increase the attendance at the Convention.

VIA THE ATCHISON, TOPEKA AND SANTA FE RAILWAY

Lv. Chicago, 10:30 P. M., July 9, Ar. Kansas City, 10:30 A. M., July 10
Lv. Kansas City, 10:45 A. M., July 10, Ar. Newton, 3:45 P. M., July 10
Lv. Newton, 4:00 P. M., July 10, Ar. Colorado Springs, 7:00 A. M., July 11
Lv. Colorado Springs, 5:50 P. M., July 11, Ar. Santa Fe, 9:15 A. M., July 12
Lv. Santa Fe, 12:15 P. M., July 12, Ar. Albuquerque, 3:15 P. M., July 12
Lv. Aubuquerque, 4:35 P. M., July 12, Ar. Grand Canyon, 7:00 A. M., July 13
Lv. Grand Canyon, 7:25 P. M., July 13, Ar. Los Angeles, 2:35 P. M., July 14

For those desiring to travel via Colorado Springs, the Royal Gorge and Salt Lake City direct to Los Angeles or from Salt Lake City via San Francisco to Los Angeles, the following schedule has been arranged:

SPECIAL TRAIN SCHEDULE VIA THE CHICAGO AND NORTH WESTERN RAILWAY AND UNION PACIFIC SYSTEM

(Sleepers ready 9:00 P. M. Sunday, July 9).

Lv. Chicago 12:15 A. M. (C. T.) Sunday, July 9, C. & N. W.
Ar. Omaha 1:45 P. M. (C. T.) Monday, July 10, C. &. N. W.
Lv. Omaha 2:00 P. M. (C. T.) Monday July 10, Union Pacific
Ar. Denver 4:00 A. M. (M. T.) Tuesday, July 11, Union Pacific

Lv. Denver 4:30 A. M. (M. T.) Tuesday, July 11, D. & R. G.
Ar. Colorado Srpings 7:00 A. M. (M. T.) Tuesday, July 11, D. & R. G.
(Day and evening to be spent in and around Colorado Springs, visit to Pikes Peak, Crystal Park Auto Trip, Cripple Creek, Manitou, Garden of the Gods, Cave of the Winds, etc.)
(Sleepers ready for occupancy 9:30 P. M.)
Lv. Colorado Springs 4:00 A. M. (M. T.) Wednesday, July 12, D. & R. G.
Ar. Royal Gorge 7:00 A. M. (M. T.) Wednesday July 12, D. & R. G.
Ar. Tennessee Pass 12:00 N'N (M. T.) Wednesday, July 12, D. & R. G.
Ar. Glenwood Springs 3:15 P. M. (M. T.) Wednesday, July 12, D. & R. G.
Ar. Salt Lake City 7:00 A. M. (M. T.) Thursday, July 13, D. & R. G.
(6 hours and 50 minutes stop-over at Salt Lake City)
Lv. Salt Lake City 2:50 P. M. (P. T.) Thursday, July 13, Union Pacific
Ar. Los Angeles 2:40 P. M. (P. T.) Friday, July 14, Union Pacific

Those who wish to travel via San Francisco will have four hours and forty minutes in Salt Lake City, leaving via Western Pacific Railroad on the following schedule for San Francisco:

Lv. Salt Lake City 12:40 P. M. (P. T.) Thursday, July 13, Western Pacific
Ar. Feather River Canyon 7:00 A. M. (P. T.) Friday, July 14, Western Pacific
Ar. San Francisco 6:00 P. M. (P. T.) Friday, July 14, Western Pacific

If rail line is desired between San Francisco and Los Angeles, the following train may be used:

Lv. San Francisco 8:00 P. M. (P. T.) Friday, July 14, Southern Pacific
Ar. Los Angeles 9:30 A. M. (P. T.) Saturday, July 15, Southern Pacific

Advance information, descriptive literature and sleeping-car reservations may be secured by addressing H. G. Van Winkle, General Agent, Chicago and North Western Railway, 148 South Clark Street, Chicago, Ill.

Members of the Association who desire to return thru Canada and the Canadian Pacific Rockies should ally themselves with the party in charge of D. C. Bacon.

The proposed itinerary from Seattle to Chicago is as follows:

Lv. Seattle, Saturday, July 29, 9:00 A. M., C.P.R. Princess Steamer.

Ar. Victoria, Saturday, July 29, 1:15 P. M., C.P.R. Princess Steamer.

Lv. Victoria, Saturday, July 29, 2:15 P. M., C. P. R. Princess Steamer.

Ar. Vancouver, Saturday, July 29, 7:00 P. M., C.P.R. Princess Steamer.

One-half mile from station to Hotel Vancouver, transfer twenty-five cents each way.

Lv. Vancouver, Sunday, July 30, 8:30 A. M., Can. Pac. Ry., Train No. 4.

Daylight trip thru Fraser, Thompson and Black Canyons.

Ar. Sicamous, Sunday, July 30, 10:28 P. M., Can. Pac. Ry., Train No. 4.

Stop over night at Hotel Sicamous to complete daylight trip thru mountains and canyons to Glacier.

Lv. Sicamous, Monday, July 31, 9:10 A. M., Can. Pac. Ry., Train No. 14.

Daylight trip thru the Selkirks and Canadian Pacific Rockies.

Ar. Glacier, Monday, July 31, 1:40 P. M., Can. Pac. Ry., Train No. 14.

Lv. Glacier, Tuesday, August 1, 3:00 A. M., Can. Pac. Ry., Train No. 14.

Ar. Lake Louise, Tuesday, August 1, 10:00 A. M., Can. Pac. Ry., Train No. 14.

Three and one-half miles from station to Chateau, Lake Louise by Narrow Gauge Railway, transfer fifty cents each way.

Lv. Lake Louise, Tuesday, August 1, 8:10 P. M., Can. Pac. Ry., Train No. 14.

Ar. Banff, Tuesday, August 1, 9:12 P. M., Can. Pac. Ry., Train No. 14.

One and one-half miles from station to Banff Springs Hotel, transfer fifty cents each way, or a delightful walk.

Lv. Banff, Wednesday, August 2, 9:22 P. M., Can. Pac. Ry., Train No. 14.

Ar. Minneapolis, Friday, August 4, 4:45 P. M., Soo Line, Train No. 108.

Ar. St. Paul, Friday, August 4, 5:30 P. M., Soo Line, Train No. 108.

Three hours to visit Twin Cities.

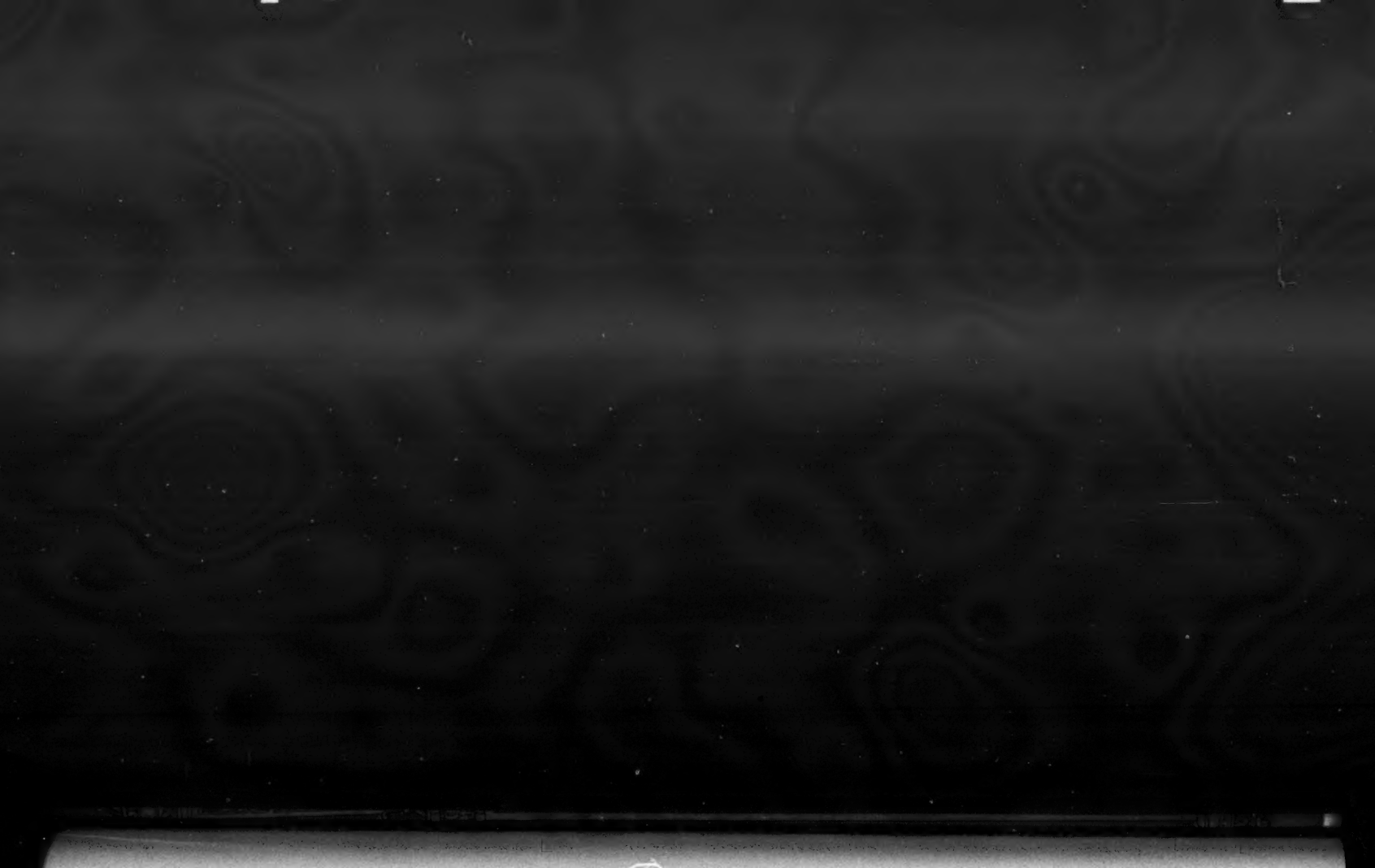
Lv. Minneapolis, Friday, August 4, 7:05 P. M., Soo Line, Train No. 4.

Lv. St. Paul, Friday, August 4, 7:40 P. M., Soo Line, Train No. 4.

Ar. Chicago, Saturday, August 5, 9:15 A. M., Soo Line, Train No. 4.

It is our intention to travel via above mentioned route in special standard sleeping cars. Arrangements will be made with the Canadian Pacific Railway to occupy the cars while at the different stops in the Canadian Rockies. Sleeping car rates Vancouver to Chicago will be as follows:

Upper berth	$ 25.65
Lower berth	32.03
Drawing room	113.30

Meals at the Canadian Pacific Hotels are a la carte service. At Banff and Lake Louise table d' hotel, $1.50 luncheon, $2.00 dinner.

D. C. Bacon, *Chairman*
31 N. State St., Chicago, Ill.